THE RETINAL CIRCULATION

IN THE NORMAL AND PATHOLOGICAL STATE

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PREFACE

It will, perhaps, be surprising to find my name at the foot of this preface; but, although I am not much of an expert in ophthalmology, the subject is by no means wholly indifferent to me, and I owe it a great deal.

It was more than twenty years ago; I was concerned at that time with clinical manifestations of hypertension, and I had been struck with the singularity of certain of these manifestations—fugacious pareses, aphasia, amaurosis, sudden and transitory, often observed in eclampsia and lead colic. Their occurrence in the course of ailments so different in appearance was a matter of surprise, but at that day nobody was embarrassed by them, and as we all recall, "hysteria" was the answer to everything. This did not satisfy me, and I rather thought that these symptoms were due to a spasm of the vessels and to hypertension, a condition common to the two diseases in question. But how to prove it?

Ophthalmology came to my aid. Physicians who had examined the eye fundus of eclamptic women, or of subjects in the crisis of acute plumbism and suddenly attacked with blindness, had not demonstrated any lesion; they had merely noted a shrinking of the vessels, with ischemia of the disc, which disappeared as if by magic, with the return of the vision to normal. Here was the confirmation of what I had propounded. It made me very glad, and from that moment I perceived the interest which a systematic study of the retinal circulation would have in diseases of the cardiovascular system, not doubting that our ophthalmological brethren would some day utilize it.

When, a long time afterward, Bailliart came to me to take part in his researches on the measurement of the pressure in the ocular vessels, I joined him with alacrity, and undertook to follow up these investigations in my clinical service with my pupils. He taught me, on that occasion, or rather to respect my *amour propre,* he re-taught me, how to manipulate the ophthalmoscope.

However, this was not a sufficient motive for his asking me to introduce his book to the medical public. He must have had another, better motive, and I think it is this:

The domain of medicine is so broad that it has been necessary to divide it into special territories, whose number increases continually. There is an advantage in this, namely, to concentrate the efforts of a few upon one restricted subject, thus enriching it with ideas which would have escaped physicians who are not specialists. But this advantage has its counterpart. In studying always the same subject, one ends by losing contact with the rest of medicine. Now, as Professor Richet has well said, "It is at the borderlines of science that the greatest discoveries are made." We shut ourselves out from them if we erect a kind of barrier between them. Moreover, the specialist, if he stays in his own office, subjects himself to the risk of only seeing lesions in their full development, when they have produced the more or less serious disorders for which the patient comes to him. He runs the risk of ignoring the mode of onset, or the initial phase, which, however, is so fruitful in information and in order that he may know them he must from time to time go back to the very fundamentals of medicine, the source of fruitful inspiration.

Bailliart realized this, and this is why he asked, not one of the masters in ophthalmology, but a physician,

to write this preface. In coming to me, who have devoted myself to the study of the pathology of the heart and the vessels, he does so most likely with the object of indicating that disorders of the retinal circulation have intimate relations with those of the general circulation, and that one cannot, under pain of making his work sterile, consider them in an isolated aspect. These are, in my opinion at least, the reasons which dictated his choice.

It is possible, of course, that I deceive myself, and that Bailliart simply wished to express his regard for me. In that case, I am only the more appreciative. He knows that he already has mine, and in what esteem I hold him. Besides, the task which he has entrusted to me is easy and almost superfluous, his work recommending itself to the attention of ophthalmologists and physicians by its own originality, its exhaustiveness, and its clarity. He has nothing to thank me for; it is I who am obligated to him. This book, in point of fact, seems to me to be assured of such success that it is quite capable, after having yielded full honor to its author, of sparing a little, out of its overflow, for its foster-parent.

H. VAQUEZ.

CONTENTS

INTRODUCTION

When an observer looks for the first time at the fundus of the eye through an ophthalmoscope, when he succeeds in obtaining an exact image, it is neither the retina which he sees visualized before him, nor the optic nerve of which it is the materialization, but the retinal circulation which attracts his attention. He is astonished at recognizing so distinctly arteries and veins, at being able to follow them so easily to their finest ramifications, and to see the circulation taking place through their walls.

The interest which this circulation has for him grows still greater when he recalls that the retinal vessels are in truth the visualization of the cerebral vessels, having a common origin with them, the division of the internal carotid stem. It then occurs to him that alterations of the one should affect the others, and that the condition of the retinal vessels undoubtedly reflects, in most cases, that of the cerebral vessels. Is it necessary to suggest the importance of the cerebral circulation? Every disorder, however slight, which attacks it immediately brings in its wake more or less serious, often fatal, symptoms. The function of the cerebral cell ceases with the circulation.

The same is true of the retinal cell. Ophthalmologists well know the blindness, sudden and complete, which supervenes when the retinal circulation is suspended. Embolism of the central retinal artery, compression of the globe, mechanically causing this arrest of the circulation, rapidly bring about an equally definite and progressive suspension of the retinal function.

We are fortunate in being able to observe directly, thanks to our modern ophthalmoscopic methods, and to more and more perfected instruments and technics, the retinal circulation, and thus to view the magnified retinal vessels in all their details, both in the normal and in the pathological state. Let us seek to apply to their study the methods which the cardiologists employ in the case of the arterial and venous trunks. Let us adopt their technic to this special examination. We shall the better acquaint ourselves with the retinal circulation. Perhaps, also, after having borrowed from physiology and cardiology their methods, to apply them to the study of the ocular fundus, profiting by the exceptionally favorable conditions in which we find ourselves for this examination, we may be able, in our turn, to furnish them with facts of which they stand in need.

In writing this book I have thought often of my loyal friend and assistant, Dr. Blutel, who died in my ambulance a few days before the armistice. It was with him that I first discussed some of the subjects to be dealt with later. His clear and penetrating spirit, his always alert intelligence, were often my guide and help. I owe him this remembrance.

In the first part of this book I treat of the anatomy, and above all of the physiology, of the retinal circulation, giving an important place to the study of local vascular pressure and its determination by means of the technic which I have recently suggested.

The second part deals with disorders of the retinal circulatory function. Notably will be found there a description of local arterial hypertension and its consequences.

Finally, the third part is devoted to lesions of the retinal vessels, and to a study of the retinal circulation in certain pathological conditions.

CHAPTER I

THE ANATOMY OF THE RETINAL CIRCULATION

The retinal circulation presents certain peculiarities upon which it is meet to lay stress.

It is an important consideration that the circulation of the eye, like that of the brain, occurs in a closed cavity. Though the ocular tunics are to some extent elastic, changes in intra-ocular tension are insufficient to affect them, so that variations in the systolic-diastolic retinal blood pressure modify but the intra-ocular tension, and vice-versa.

The analogy between the ocular and the cerebral circulation is quite close. A rise in intra-cranial tension tends to be opposed by a passive diminution in caliber of the venous sinuses. Similarly in the eye, the large vorticose veins act like the cranial sinuses to moderate any change in intra-ocular tension. Normally the variations in tension are indeed very slight.

It is obviously advantageous for the delicate tissues of the eye and the brain that their cir-

culation be maintained under quite constant conditions. The functions of a limb are not disturbed by the brusk alterations in pressure to which its artery is subject, but such variations as are permissible in the brachial artery would not be without danger to highly organized nervous structures.

The retinal arteries, like those of the brain and kidney, are terminal. Their only communication with each other is in the capillary bed. The obliteration of an arterial branch means the complete and permanent anemia of the area supplied.

The retinal veins are for the main part independent of each other. However, there are some anastomoses between the venules in the anterior portion of the retina, not far from the ora serrata.

The Retinal Arteries

The circulation of the retina depends almost entirely upon the a. centralis retinae. This artery arises from the ophthalmic artery on the entrance of the latter in the orbit and at first goes along the lateral surface of the optic nerve, to which it is attached by dura mater. The artery then penetrates into the interior of the nerve at a distance of 10-15 mm. from the eyeball and follows thereafter in its central axis to the optic papilla, where it bifurcates.

The artery may divide and redivide in the

optic nerve before it has reached the sclera, though generally it does not bifurcate until the papilla is reached. The ascending and descend-

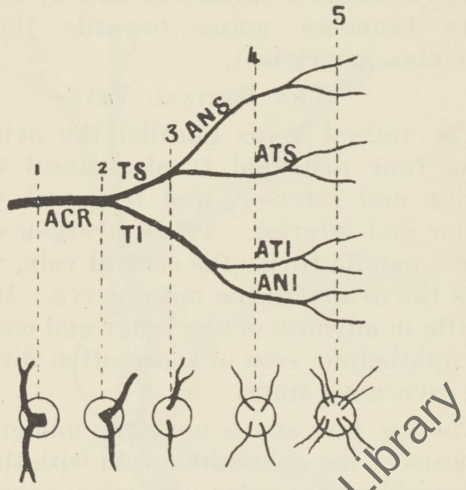

Fig. 1. The central retinal artery and its branches. Various opthalmoscopic appearances, according to the location of the points of bifurcation in relation to the surface of the papilla.

(Schema of Hollet.)

1 and 2. The central artery bifurcates at the surface of the disc. 3. Bifurcation behind the lamina cribrosa, invisible with the ophthalmoscope. 4. Two successive bifurcations are made behind the lamina cribrosa, and the retinal arteries each divide into two branches where they penetrate the eye, a very exceptional case.

(Poirier and Charpy, Human Anatomy.)

ing branches formed give rise in turn to nasal and temporal divisions. The ophthalmoscope

presents a varying picture of these tributaries according to the place at which division occurs.

Not infrequently one meets a branch directed directly forward (arteriola media) and one or more branches going towards the macula (macular arterioles).

The Retinal Veins

The retinal veins parallel the arteries and form four principal trunks: nasal veins, superior and inferior, and temporal veins, superior and inferior. The convergence of these at the papilla forms the central vein, which follows the artery in the optic nerve. It emerges a little in advance of the latter and empties into the ophthalmic vein or more often directly into the cavernous sinus.

Charpy[1] lays stress upon the numerous anastomoses of the ophthalmic vein with the frontal, supraorbital, angular, inferior cerebral and other veins. Any disorder in this area drained by the ophthalmic vein is capable of affecting retinal circulation. On the other hand, blood-letting from incisions made at the lateral angle of the eye may thus exert a favorable action in the treatment of certain ocular affections.

Cilio-retinal Vessels

Occasionally there arises from the temporal border of the disc one or more arterioles, called cilio-retinal, whose origin is independent of the a. centralis retinæ. These vessels when

present supply with blood a small district of the retina lying between the papilla and macula.

Jackson in a thousand eyes examined oph-

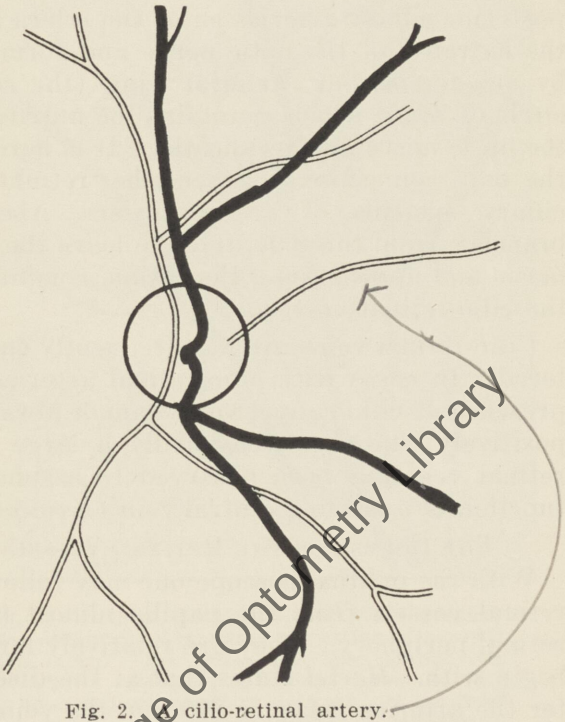

Fig. 2. A cilio-retinal artery.

thalmoscopically found cilio-retinal arteries present in one-fifth of the number. In a case of *Mulle's* it was demonstrated that the cilio-

retinal artery observed in the ophthalmoscope did not arise from the central artery.

The presence of these vessels is thus explained: Some branches (2-4) from the short posterior ciliary arteries enter the sclera about the entrance of the optic nerve and form in it by anastomosis an arterial ring (the scleral circle of Zinn) which maintains the nutrition of the optic nerve and its sheaths. It is here that the only connection between the retinal and ciliary systems of vessels exists. Aberrant branches from this scleral circle leave the optic nerve and appear upon the retina, constituting the cilio-retinal vessels.

Cilio-retinal veins are less frequently encountered. In cases with cilio-retinal arteries, the presence of cilio-retinal veins cannot always be positively stated. Occasionally a large cilio-retinal vein has been observed to assume the function of a missing central vein (*Kraupa*[2]).

The Course of the Retinal Vessels

With the ophthalmoscope one may follow the retinal vessels from the papilla almost to the retinal periphery. They are relatively large to begin with. *Herzel* states that at the disc center the arteries measure 210 mu, the veins 245 mu, but at the extreme periphery near the ora serrata they are almost invisible.

Rochon Duvigneaud has pointed out an interesting difference between the distribution of

nerve fibers in the retina and that of the retina vessels. The decussation of one-half of the

Fig. 3. Retinal circulation. Magtot's preparation, obtained by injecting the retina of a newly born infant. The retina has been spread out, and shows arterial and venous branches with the intermediary capillary network. In the inset is a venous arborization with the capillaries. (Morax, Glaucoma and the Glaucomatous.)

optic nerve divides the retina neurologically into lateral and medial halves, while the primary bifurcation of the retinal vessels into

ascending and descending branches divides the retina from the circulatory standpoint into superior and inferior divisions.

Though the middle of the fovea is devoid of vessels, about the macula is an extremely rich capillary supply. The temporal arteries, superior and inferior, describe arcs above and below this area, and from these fine ramifications are directed towards the macula (superior and inferior macular arteries). There are also the macular arteries that proceed directly from the disc and are derived from the central artery or from one of its primary branches, and the cilio-retinal artery when present likewise supplies this area.

The course of both retinal arteries and veins, particularly the veins, is more or less tortuous, and they cross each other quite frequently. Hardly ever do artery and vein proceed side by side; the juxtaposition would probably arrest too many luminous rays.

The larger retinal vessels lie upon the nerve fiber layer and are only separated from the vitreous by the hyaloid membrane. The retinal capillaries alone are in the deeper layers of the retina.

The Retinal Capillaries

Along their entire course the retinal arteries give rise to tiny branches which penetrate to

the external plexiform layer, but never beyond. Two capillary beds are formed. The external network is closely meshed and situated in the inner nuclear layer; the internal network is much looser and placed in the nerve-and-ganglion stratum. The former is associated with the retinal veins; the latter with the retinal arteries.

The retinal vascular system then is entirely confined to the internal portion of the retina. The external layers, composed of the rods and cones and the visual cells, are devoid of vessels. Since the macula is composed only of these external layers (the neuro-epithelial part of the retina), we find this spot, as might be expected, entirely non-vascular. The middle of the fovea appears like a hole in the dense capillary net about it. The dimensions of this gap are, however, not extensive, varying from a diameter of .2 to .8 mm., occasionally .13 mm. (Dimmer). The fovea has behind it the chorio-capillaries, here particularly well developed, which to a considerable extent takes care of the nutrition of this non-vascular area.

The Structure of the Retinal Vessels

The arteries of the retina have the usual coats, but the veins resemble capillaries in structure, their walls consisting of a single layer of endothelial cells without any muscular tissue (Schäfer). Outside the endothelial layer

is a space both in veins and capillaries, bounded externally by a second endothelial layer. In addition, the veins have outside this a layer composed of a peculiar retiform tissue. These perivascular spaces have been filled with colored fluid injected under the sheath which the optic nerve derives from the pia mater (Schwalbe). These spaces hence are not lymphatic, but filled with cerebro-spinal fluid.

The Ophthalmoscopic Appearance of the Retinal Vessels

With the ophthalmoscope the arteries and veins of the retina are easily distinguished. The arteries are brighter red, narrower, and run a straighter course; the veins are darker, of greater caliber and more tortuous. On the arteries, there is a prominent shining streak along the central axis. This reflex streak along the veins is much thinner and much less noticeable, occupying but 1/14 of their diameter (Adams).

A venous pulse at the spot where the vein first appears upon the papilla is a frequent physiological occurrence; an arterial pulse is seen, however, only under pathological conditions.

In children the retina has a luster like that of watered silk. The vivid reflexes which shift with every movement of the mirror are especially pronounced along the vessels. The

tendency of the retinal vessels to project slightly in the vitreous produces reflecting surfaces, and because the juvenile retina fits but loosely against the subjacent coats, these reflexes are accentuated in infants and young children.

All these reflexes are particularly brilliant in ophthalmoscopy with red-free light. This illumination makes the vessels appear black on a green background. They are thus more distinctly seen and their ramifications can be followed much further than ordinarily.

The Embryology of the Retinal Vessels

The retinal vessels appear rather late in ocular development. The embryonic eye begins with a peripheral vascular network, the hyaloid plexus (vasa hyaloidea propria), whose main stem becomes enclosed in the anterior portion of the optic nerve. This "central artery of the optic nerve" (later of the retina) continues as the a. hyaloidea through the vitreous to the posterior pole of the lens. On arriving at the lens the artery divides into numerous ramifications that cover its posterior surface. At the lens margin these unite with the anterior extremities of the vasa hyaloidea propria and form a dense plexus about the lens border (tunica vasculaso lentis).

In the 10 cm. fetus, the central artery of the optic nerve is already surrounded by a venous

plexus with two leading trunks, which on leaving the nerve unite into one. The veins do not accompany the artery into the vitreous, but stop shortly after reaching the disc. At this stage numerous vascular buds on both arteries and veins are visible near the papilla. In the fetus of 12-13 cm. these buds become permeable, constituting the earliest retinal vessels. These develop gradually till little by little they cover the retina. In the six-month fetus the ora serrata has not yet been reached. As the retinal circulation extends, the hyaloid system previously present undergoes degeneration.

With the progress of fetal development, the central vein becomes formed nearer and nearer the disc, till in the fetus of 22-24 cm. it appears at about the disc level.

The retinal vessels are on the nerve fiber layer and do not send branches to the deeper strata till comparatively late. In the fetus of 18-19 cm., ramifications have reached the ganglionic layer; in the 36 cm. fetus the internal capillary plexus has formed, and in the 42 cm. fetus the external capillary plexus.

The retinal circulation is not similarly derived in all mammalia. In man the origin of the retinal vessels is associated with an antecedent hyaloid plexus. Among many others, as in the cat, the retinal circulation is derived from the choroidal. The cilio-retinal vessels

sometimes seen illustrate this situation in a very minor way occurring in man.

The Retinal Circulation Among Animals

With one or two exceptions (Chelonia, eel) no vertebrates but mammals have blood vessels in the retina; even in some mammals the distribution is restricted to the posterior part of the eye and to the nerve fiber layer.

Lindsay Johnson distinguishes four types of retinal circulation among mammals:

1. The central artery and vein are well developed and their ramifications extend throughout the retina.

2. Numerous vessels emerge from about the disc and supply the larger portion of the retina.

3. The retinal vessels are only slightly developed.

4. Absence of retinal vessels (ophthalmoscopically).

A few details on the character of the retinal circulation in some of our domestic and laboratory animals may be of interest. In the dog and cat the circulation is only a little less developed than in man. The ruminants, the hog and the rat follow next.

In the dog the arterial supply of the retina is shared between the a. centralis retinæ and the ciliary arteries. The arteries emerge from and about the papilla already considerably subdi-

vided; the central veins, however, are quite large.

Though the retinal supply of the cat is solely ciliary, the appearance of the disc surface is not much unlike that of man. Three to four main trunks (arteries and veins) leave the periphery of the papilla and are extensively distributed.

The retinal circulation of horse and rabbit are quite rudimentary. In the horse a number of vessels leave the disc, but extend only a short distance. Just below the papilla there is even a trapezoid zone entirely devoid of vessels.

In the rabbit, a band of medullated nerve fibers extends horizontally on each side of the disc. On each of these bands is an artery and a vein. Their branches are very fine and never pass beyond the zone of medullated fibers.

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CHAPTER II

THE PHYSIOLOGY OF THE RETINAL CIRCULATION

The retinal circulation has many peculiar characteristics; it is displayed to inspection from the arrival of the red arterial blood to the exit of the dark venous; the velocity of its current can be measured; the arterial and venous blood-pressure noted; vasomomotor phenomena are directly visible, and, as has already been noted, the retinal circulation like the cerebral occurs in a closed cavity in a milieu of almost constant tension.

The Circulation and the Retinal Function

The visual function is absolutely dependent on retinal circulation. When the central artery is obliterated by a thrombus, embolus or spasm, forthwith total blindness results, and if but a branch artery is affected, in the sector corresponding to that branch, blindness is produced. We can try this experiment: With one eye closed, a finger exerts gradually increasing pressure on the other open eye. When the compression is strong enough to stop the flow in

the central artery, vision becomes obscured and then disappears. The sensitivity of the visual cell to circulatory observation is only equalled by the cerebral tissue.

The Pressure in the A. Centralis Retinae

The Retinal Pulse.—The retinal arteries normally appear immobile. The pulse excursion in vessels of such small caliber is too slight under physiological conditions to be detected ordinarily. However, *Kummel*[1] and *De Speyr*[2] find that with the magnifying ophthalmoscope of Gullstrand this phenomenon can almost constantly be observed.

In certain pathological conditions, however, an arterial pulse occurs that is revealed to the ordinary ophthalmoscope. Such cases are found in glaucoma, syncope, severe anemia and aortic regurgitation.

If while looking at the fundus with the ophthalmoscope the observer exerts gently increasing pressure on the globe, an arterial pulse will appear. This induced pulse is particularly marked in juveniles. In the adult the pulsation induced is less extensive, but in all cases the pulse can be noted, though in the aged it may only be observable at one point. This pulsation can be seen in indirect ophthalmoscopy, but the details are much better studied in the direct method.

As the pressure on the globe increases, so does the amplitude of pulsation to a certain

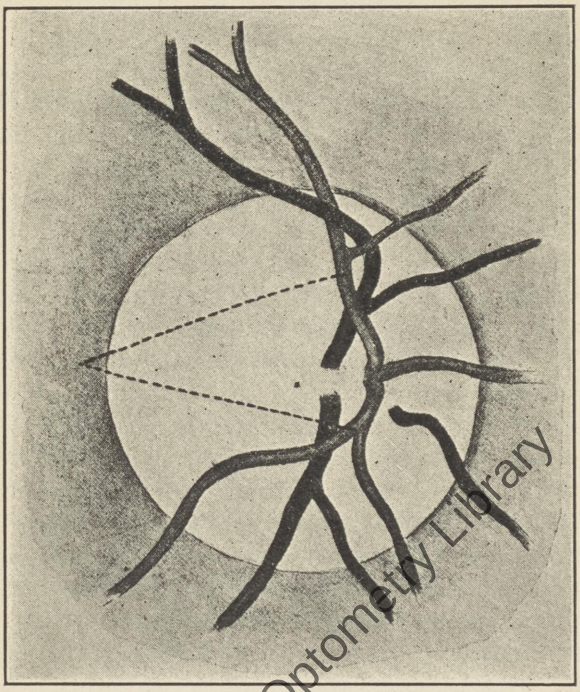

Fig. 4. The retinal circulation on the disc. Between the branches of the vein are the points where the vascular pulsations are conveniently seen.

point, and then grows less and disappears. If the pressure is further continued, suddenly the artery becomes effaced and, blended with the

tissue anemia about it, is no longer visible.

The mechanism of the arterial pulse, whether spontaneous or induced, is based on the same principle. As intra-ocular tension increases, the lumen of the artery is diminished, and when the intra-ocular tension is equal to the diastolic blood pressure within the artery, one neutralizes the other. The diameter of the artery then is that assumed by a vessel containing blood under no pressure and kept patent only by the stiffness of its walls. The pulse wave, finding this portion of the artery thus completely relaxed, causes a maximum distention of its walls, and a correspondingly large pulse excursion. If the intra-ocular tension is increased beyond the diastolic pressure, it not only neutralizes the latter, but overcomes the stiffness of the arterial wall. In this case the pulse wave must expend part of its energy in overcoming the excess pressure on the outside, and accordingly the movements of the walls with the pulse wave are less extensive in proportion to the excess of pressure on the outside.[3]

Normally the diastolic pressure in the central artery is greater than the intra-ocular tension. Hence to produce a visible arterial pulse we must increase this tension by pressure on the globe. Pathologically, the intra-ocular tension and the diastolic pressure may become more or

less equalized in two ways: in glaucoma the intra-ocular tension is increased; in syncope, anemia and aortic regurgitation, the diastolic pressure is reduced.

Determination of the Arterial Retinal Blood-Pressure.—The visible arterial pulse appears at the moment that the ocular tension becomes equal to the local diastolic pressure, and disappears as soon as the local systolic pressure is exceeded.

In 1909 the author[4] noted that in patients suffering from hypertension it required quite strong finger pressure on the globe to cause the disappearance of the arterial pulse. *Melville Black*[5] in 1911 and *Deyl*[6] in 1912 agreed on the importance of this finding. In 1914 Thomas Henderson invented the first registering instrument for compression of the eyeball. With this instrument pressure was applied on the lid, and though designed for the study of retinal blood pressure, the findings were quite inaccurate.

Von Schulten[7] tried to study the question in the rabbit by intra-ocular manometry. The difficulties of the experiment, however, are such as to make his conclusions wholly unreliable. In 1917 *Priestley-Smith*[8] began an extensive study of retinal blood-pressure, but unfortunately used Von Schulten's data.

Technic. (a) **The Use of the Dynamometer.**—The author, in 1917, designed an instrument

for compression of the eyeball based on the tension of a spring, which was inspired by the old sphygmomanometer of *Bloch-Verdin*. The instrument was named an ophthalmo-dynamometer. The graduations are in grams water, and in the present model, the graduated rod measures 15-150 grams. In an alternative model the rod is replaced by a dial on which a pointer registers the pressure.

The terminal disc which serves as the compressing surface is very slightly convex. Though a concave disc would be more adapted to the surface of the eyeball, the results with it tend to be more unreliable, for when the observer thinks its entire surface is compressing the eyeball, it may be that but an edge is against the globe. This, of course, creates an error, for by the laws of hydrostatics the pressure transmitted to a liquid is proportional to the area compressed.

Moreover, should the instrument slip, the slightly convex disc is unlikely to do the cornea any harm.

The compression should be made directly upon the eyeball. Pressure made through the upper lid gives but approximate values, as the resistance of the lid tissues varies in different individuals. Nevertheless, this method can be recommended to novices as it is well adapted for purposes of training.

In exact work the instrument must be applied directly against the conjunctiva. The most suitable point is the outer portion of the eyeball, a little posterior to the insertion of the external rectus muscle. The instillation of two drops of holocaine or of 2 per cent cocaine facilitates the examination. The procedure is generally quite painless, only the sense of pressure being felt. It is inadvisable, however, to employ a pressure of over 150 grams. This is less than that which the patient would use in squeezing his own eyeball, and quite below that required to produce an ocula-cardiac reflex.

The instrument must be applied perpendicularly to the globe and in the horizontal plane, and kept so throughout the examination. It must not be allowed to slip above, below, or posteriorly. Otherwise, the dynamometer expends but part of its force on the eyeball, and too high readings will be given.

(b) **The Ophthalmoscopic Examination.**—With the dynamometer placed for compression, the observer concentrates on the arteries at the disc—not beyond.

The novice may perhaps find the examination with indirect ophthalmoscopy easier. Even with the magnification thus obtained, the arterial pulse will be easily recognized. However, one cannot tell with this method just

when the pulse appears, and just when it disappears. Moreover one must rely upon an assistant to make compression and give the readings, which in the obscurity involved in ophthalmoscopy is difficult. The various errors tend to make the results obtained by this method uniformly too high.

Every advantage is accordingly with direct ophthalmoscopy. Though no assistant is needed, an electric ophthalmoscope is essential, as the position of the dynamometer would interfere with the reflection of light by an ophthalmoscopic mirror.

On the dynamometer is a raised ring which the observer grasps between the thumb and index finger of the left hand. After the instrument is placed in position, the free fingers rest against the temple and steady the hand. Pressure is commenced gently. As soon as the pulse appears, the light from the ophthalmoscope is thrown on the graduated rod and the reading noted. Pressure is then continued till the pulse disappears, when a second reading is made.

The movement of the head necessary to note the findings, after a little practice, will occasion no difficulty. Still with a free finger one could hold the rod in the registered position and bring the instrument to the light. The procedure is simplified moreover in the latest

dial dynamometer in which the pointer automatically stops when compression ceases.

(c) **Precautions.**—Theoretically it is the maximum pulsation that should denote the diastolic pressure. Practically there is so little difference between this and the first visible arterial pulse that, for the retinal artery, the latter can be considered a proper criterion.

Not to be misled, however, in the appearance of the first pulse, the observer must assure himself that a series of regular pulsations follow it. Similarly, the systolic pressure should be checked up by noting the recurrence of a true pulse on slightly diminishing the pressure.

Though the compression should proceed gently, the readings must be determined without undue delay, otherwise changes in intra-ocular tension tend to take place and too high readings may result.

2. **The Dynamometer and Intra-Ocular Tension.**—The dynamometer readings indicate but the gram pressure applied to the eyeball, but do not give the pressure transmitted to the retinal arteries. To learn this, we must study just how intra-ocular tension is modified by outside pressure. A simple addition of dynamometer pressure and intra-ocular tension would give a false value owing to physiological changes that come into play. Two lines of investigation have been pursued to determine the desired data:

Suppose, in a certain case, the dynamometer registers 25 gm. at the appearance of the arterial pulse, and 75 gm. at its disappearance. We can then measure the tension of the eyeball directly with the Schiötz tonometer under these particular conditions: that is, when the dynamometer exerts 25 gm. pressure, and again when it exerts 75 gm. pressure.

This simultaneous application of dynamometer and tonometer, however, can only be successfully accomplished on very docile patients. *Magitot* and I' determined consequently to investigate the subject experimentally in the anesthetized cat. A needle connected with a manometer was introduced into the anterior chamber, and readings taken as the pressure with the dynamometer was varied. Numerous cats' eyes were thus investigated, covering quite a range in initial intra-ocular tension. On the adjoined graph is a record of the findings. The solid lines represent results experimentally obtained. The dotted lines are the curves that may be assumed on this basis in cases of initial tension with higher values. Employing this table, one but needs to know the initial intra-ocular tension to translate the dynamometer readings at the appearance and disappearance of the arterial pulse into terms of millimeters mercury.

The values thus obtained are somewhat

higher than result when dynamometer and tonometer are simultaneously used. However,

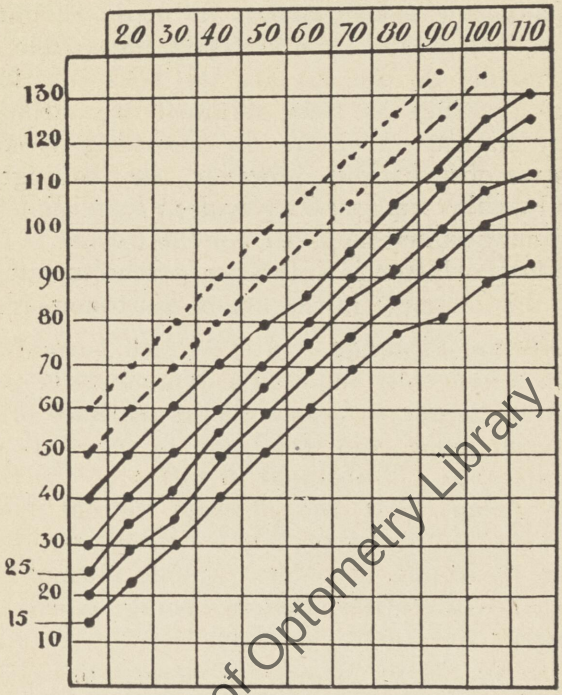

Fig. 5. Modification of the intra-ocular tension under the influence of dynamo-metric weights. (Magitot and Bailliart.)

the readings with the Schiötz tonometer are probably always a few millimeters low. The difference in the eyes of cats and men as re-

gards the elasticity of the tunics, and the volumetric capacity are also possible modifying factors. Accuracy would require that the manometric measurements be made on human eyes, but as this is manifestly impossible, experiments on the cat are the best substitute. The readings as thus obtained are probably only slightly in error. In recording dynamometric observations, however, the readings as well as the supposed equivalent in millimeters mercury should be given, for the former is fact, while the latter is inference. Schiötz himself set this example in the use of his tonometer.

Normal Findings.—In eyes with normal tension it generally requires a dynamometer pressure of 25 gm. to induce the appearance of the arterial pulse, and 60-70 gm. to cause its disappearance. Translated in terms of the table this means that the average retinal blood-pressure is: Diastolic, 30-35 mm. mercury; systolic, 65-70 mm. mercury. Among normal individuals—all other factors being equal—the findings are fairly uniform.

In the early efforts to determine retinal blood-pressure previously referred to, *Von Schulten* estimated the systolic pressure as 90-100 mm. mercury; and *Henderson*[10] the diastolic as 15-25 mm. above the intra-ocular tension.

The arterial pulse is not seen except on the disc probably because beyond the papilla the

vessels are more or less embedded in retinal tissue. On this account, it is to be regretted, the blood-pressure in the retinal arteries beyond the disc cannot be measured.

When cilio-retinal vessels exist their pressure is generally equal to that in the retinal arteries, though their caliber appears smaller. Occasionally, however, there may be as much as 5 mm. mercury difference between them.

Physiological Variations.—The retinal, like the systemic, blood-pressure is somewhat lower in the young and higher in the aged. Before a meal the readings are lower than after. The effect of respiration is inappreciable. Change of position likewise affects retinal blood-pressure but little.

In a study of the cerebral circulation *Francois-Franck*[12] similarly finds that because of compensating factors position has not the influence that conclusions based simply on considerations of gravity would lead one to expect.

It has been difficult to determine the effect of accommodation on the retinal blood-pressure, mainly because we have been unable to determine what changes in intra-ocular tension occur in the process. The dynamometer generally indicates an elevation of some mm. mercury.

The Relation Between Systemic and Retinal Blood-Pressure.—Under physiological condi-

tions, the pressure in the retinal arteries is entirely dependent on systemic blood-pressure and follows its variations. Knowing the systemic blood-pressure one can generally safely predict the retinal blood-pressure, and vice versa. Multiplying the systemic diastolic pressure by .45 will give the retinal diastolic pressure. If the reciprocal relation shows a variation of more than 5-10 mm. mercury from that observed, the lack of accord should be noted and investigated.

There is a somewhat less constant relationship between the systolic pressures, but multiplying the systemic systolic pressure by the coefficient .54 generally gives a good approximation to the retinal systolic blood-pressure.

The diastolic pressure in the retinal artery is already almost on the capillary level. The systolic pressure has not fallen proportionately quite so much for to it belongs the duty of overcoming the great capillary resistance; and in this task its energy will be quickly spent. In the capillary bed itself there is no pulse, and diastolic and systolic pressures become one.

To the physiologist and the student of the problems of circulation, the retina offers a unique opportunity to observe pressure conditions in the arterioles. It is the only part of the peripheral circulation that can be readily and accurately studied.

Another Procedure for the Determination of the Blood-Pressure of the A. Centralis Retinæ. —Bliedung[15] has devised a sphygmomanometric method for ascertaining the retinal blood-pressure. He encloses the eye in a partly transparent, airtight capsule. The eye is observed through the window with an ophthalmoscope, while the pressure within the capsule is increased till the arterial pulsation appears. The increase of pressure is then continued until the induced pulsation is suppressed.

Bliedung's method gives results notably higher than mine. He figures the systolic pressure, 96-112 mm. mercury; the diastolic pressure, 64-74 mm. mercury. The following considerations explain the reason for his values: First, Bliedung has added his sphygmomanometric reading to the intra-ocular tension, and gives the result as the blood-pressure of the central artery. But the actual force expended on the retinal artery, though a resultant of these factors, cannot be computed by their simple addition. Secondly, not all of the pressure in the air-tight capsule is effective on the eyeball. Part of it is spent in pushing the globe back in the orbit.

Studies on the blood-pressure of the temporal artery by this method[16], for which it is well suited (and which I had thus employed for some years previously) gives an average meas-

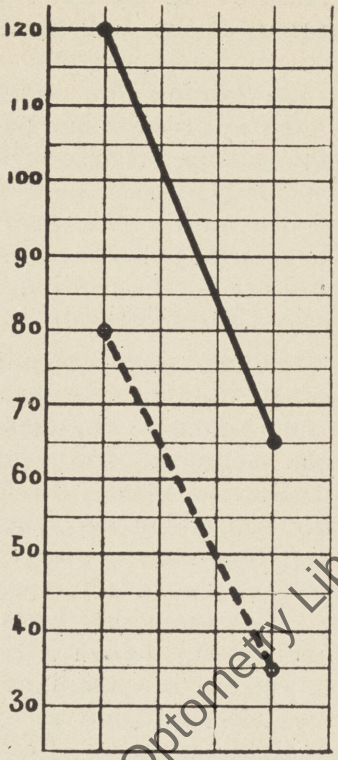

Fig. 6. Fall of arterial pressure in the brachial artery and the central retinal artery.

urement of 30 mm. mercury diastolic, 60-70 mm. systolic. The error in Bliedung's values is hence manifest, as it is inconceivable that the pressure in the central artery of the retina

should exceed that of the temporal.

Bliedung's values are almost equal to the pressure findings on the brachial artery. How much lower the actual findings are is demonstrated conclusively by certain clinical studies. In cases where a spontaneous arterial pulse exists, it requires no special technique to learn the value of the diastolic pressure. In these cases the diastolic pressure of the central artery must be in equilibrium with the intra-ocular tension, and the tonometric determination suffices to give the desired information. In aortic insufficiency a spontaneous pulse is often noted with an intra-ocular tension of 15 mm. mercury. In glaucoma, the intra-ocular tension need not be extraordinarily high for the spontaneous arterial pulse to appear. I have found that the average intra-ocular tension of the cases in which a spontaneous pulse is seen is 55 mm. mercury.

Seidel[14] measured the blood-pressure in the anterior ciliary arteries by a method similar to that used by Bliedung for his determinations of the pressure in the retinal artery. He found the diastolic pressure was 30-45 mm. mercury; systolic, 55-75 mm. These figures are on the same order as those of LePlat for the pressure of the vessels of the iris. Using my dynamometer, he determined the diastolic pressure to be 30-45 mm. mercury; systolic, 75-90.

Weiss[15] in 1923 stated that from his most recent measurements of the intra-ocular arterial pressure by the manometer, he considered the systolic pressure to be 50-70 mm. mercury. The results from these various sources are in general accord with my dynamometric findings.

Whoever employs my dynamometer should recall that the average diastolic pressure of the central artery is about 30 mm. mercury; the systolic, about 70 mm., and check his findings by these figures. The novice in the use of the instrument has difficulty of noting exactly when the arterial pulse first appears, and when it just disappears. There is consequently a tendency at first towards reading too high values.

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CHAPTER III

THE VENOUS CIRCULATION

The retinal veins are without valves. The need for them is slight, as practically no position seriously interferes with the return flow. The venous current is moreover assisted by the intra-ocular tension constantly exercised on the venous walls.

The Venous Pulse.—Pulsation in the central vein occurs physiologically in many individuals. The pulse is only seen on the papilla, for here the vessels lie wholly superficial, and without a covering of nerve fibers (Salzmann)[1]. The pulse is best observed at the point where the vein bends to descend into the physiological excavation. The beat may be well marked, or there may be an almost imperceptible flicker, or only a rhythmic change in color.

Among 66 individuals studied in 1918,[2] in 28 or 42 per cent no pulse movement was visible. However, the pulse could be induced in many of these subjects by effort, compression of the cervical vessels, forced respiratory movements, or compression of the eyeball.

In 14 or 21 per cent of the cases, venous pulsation appeared only intermittently—in some only with inspiration or expiration. After using the methods suggested above, the pulse could generally be rendered constant.

In the remaining 24 or 37 per cent, the pulsation was regular and distinct. The typical venous beat is localized and gradual, slowly developing and slowly passing. The vessel collapses for a moment, driving its contents toward center and periphery—and then recovers its caliber. Using the temporal artery as a basis for comparison, one sees the vein fill slowly in cardiac diastole, and discharge in systole. Forced inspiration makes the discharge phase longer and more powerful. With compression of the cervical vessels, the venous pulse first disappears, to reappear later with greater intensity.

Occasionally, in about 2 per cent of cases, instead of the venous wall pulsing as described, one sees rather a piston-like movement of the contained blood.

The Effects of Compression of the Eyeball on the Veins.—As pressure is applied to the eyeball the veins contract and whiten, till only a thin thread of blood maintains the circulation. With continued pressure, the vein becomes effaced, but just before this takes place the cur-

rent breaks up and assumes "a granular aspect."

The Induced Venous Pulse.—Not only are vein and artery easily distinguished, but likewise the character of their pulsations. The slow and prolonged venous pulse is quite different from the short and abrupt arterial. Occasionally, because of close contact, the arterial pulse may be transmitted to the vein. This transmitted pulse is readily recognized by noting that it parallels the arterial pulse, and has arterial rather than venous characteristics.

Though compression of the eyeball will constantly provoke an arterial pulse, it fails in one-third of the cases to induce a venous pulse. However, the usual sequence on compressing the eyeball is this: The vein first seems to retract; then the venous pulse appears; this pulsation is next arrested, and after only a few millimeters mercury increase the arterial pulse appears. Occasionally the venous pulse persists after the appearance of pulsation in the artery, the former marking the time of cardiac systole, the latter of diastole. This phenomenon may be conveniently termed "the double retinal pulse."

Should the venous pulse be physiologically present, the first effect of compression is to amplify it, especially so in the piston type of pulsation.

The Mechanism of the Venous Pulse.—The venous pulse is generally explained by assuming a rise of intra-ocular tension at systole, which momentarily effaces the walls of the central vein. *Priestly-Smith*[5], accepting the views of *Donders*, states the situation thus: The arterial wave, in dilating the intra-ocular arteries, increases the intra-ocular content, and so raises momentarily the intra-ocular pressure above that of the lowest point of venous pressure, which, of course, is in the central vein. The vein consequently is constricted at the spot where it dips down into the vascular funnel. This damming causes that portion of the vein directly adjacent to swell up. A rise of venous pressure is thus occasioned, which at the time of diastole, with its associated diminution of intra-ocular tension, readily overcomes the compression.

Morat and *Doyon*[6] make this interesting comment: Wherever a circulation occurs in a closed cavity whose walls are practically non-distensible, the increase of arterial supply with each systole favors the exit of venous blood. Such conditions occur in the brain, the eye, and the cavernous sinus. A venous pulse hence normally occurs in these organs.

According to *Haab*[7] (who follows *Helfreich*[8] in this), the venous pulse is not to be accounted for solely by changes in intra-ocular tension.

They believe that negative waves transmitted by the cavernous sinus to the veins of the orbit and fundus constitute a considerable factor in producing the rhythmic effacement of the central vein.

It will be well to consider in detail the changes that take place within and without the walls of the veins at each phase of the cardiac cycle. The venous pressure at its capillary end is fairly constant and only slightly influenced by intra-ocular tension. The pressure from the capillaries is the force that maintains the venous stream, and this becomes less and less as the veins near the papilla. And the nearer the veins approach the papilla, the more are they subject to oscillatory changes in pressure, transmitted from the jugular vein via the cavernous sinus. Auricular systole causes a backflow in the jugular—a momentary arrest of the venous stream—and a subsequent rise in venous pressure. During auricular diastole, on the other hand, there is an aspiration of blood as it were, and a corresponding fall in venous pressure. Accordingly, the pressure in the central vein passes from a minimum at the time of auricular diastole and ventricular systole to a maximum in auricular systole and ventricular diastole.

But what happens, meanwhile, outside the venous walls? The intra-ocular tension sim-

ilarly goes through an oscillatory variation, being (for reasons already stated) at its maxi-

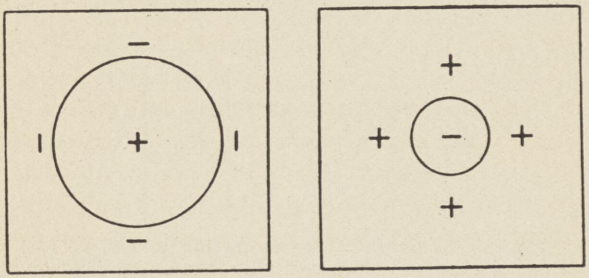

Fig. 7. Schematic representation of the venous pulse. The vein is shown suspended in the ocular cavity.

At the left, during diastole, the pressure is lower in the ocular media, and higher in the interior of the vein, thus enlarging the vein.

At the right, during systole, the pressure is raised in the ocular media and lowered in the interior of the vein, lessening its lumen.

mum in ventricular systole, and at its minimum in diastole.

Thus, at systole, since the intra-ocular tension is increased at the same time that the venous pressure is diminished, the central vein collapses; and in diastole, when the conditions are reversed, the vein becomes maximally dilated. These alternative conditions account for the venous pulse spontaneously occurring. In the accompanying diagram it is seen that then the curves for intra-ocular tension and venous pressure cross each other at each pulse beat.

If these pressure curves should not intersect, owing to the intra-ocular tension remaining ever higher or ever lower than the venous

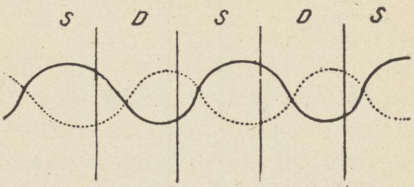

Fig. 8. Schematic representation of the retinal venous pulse. The full line shows ocular tension, the dotted line, venous pressure.

pressure, there will be no spontaneous venous pulse.

But, under these conditions, how can a venous pulse be induced?

Let us consider first *the case when the intra-ocular tension remains ever lower than the venous pressure.* To produce a venous pulse, it is but necessary to increase the intra-ocular tension by compression of the eyeball. When the tension thus modified becomes equal to the venous pressure, the vein collapses in systole, and dilates in diastole. A venous pulse, then, which is induced by ocular compression indicates that in the subject observed the intra-ocular tension is inferior to the venous pressure.

The second case is *when the intra-ocular tension remains ever higher than the venous*

pressure. The venous pressure then must be elevated by either lightly compressing the jugular vein, or by making the patient hold his breath after a strong expiration; or, the intra-ocular tension can be lowered a few milimeters for several seconds by lightly massaging the eyeball for a minute or so.[9]

Whenever, then, the venous pulse appears, an equilibrium exists between the intra-ocular tension and the venous exit pressure, the former passing the other in systole, and being in turn surpassed by the latter in diastole.

In over one-third of normal individuals a venous pulse occurs physiologically. In these cases the venous pressure at its minimum is below the intra-ocular tension, and at its maximum exceeds it, but only by a few milimeters mercury.

In 30% of the cases the venous exit pressure remains always below the intra-ocular tension. The difference is too small to efface the vein, however, and the rigidity of the venous wall maintains the circulation.

In 12% of the subjects observed the venous exit pressure remains constantly above the intra-ocular tension, though by only a small margin. The slightest compression of the globe induces the venous pulse.

When the venous pulse is intermittent, we conclude that the maximal intra-ocular tension

is so close to the minimal venous pressure that slight modifications of the latter, such as occur during the respiratory movements, cause the appearance or disappearance of the pulse.

To summarize: *The venous exit pressure in normal individuals is always near the intra-ocular tension, about which it oscillates in the phases of the cardiac cycle.* The venous pulse is not at all indicative of either arterial or venous hyper-tension.

The Values of the Venous Retinal Blood-Pressure in Normal Individuals.—*Priestly-Smith* concluded from physiological data that the venous exit pressure is only slightly above the intra-ocular tension. He cites as an interesting analogy that *Leonard Hill*, experimenting on dogs, found the pressure in the confluens sinuum (O. T. torcular Herophili) the same as that of the cerebrospinal fluid. This, in normal individuals (according to Claude's manometer), oscillates about 10 mm. mercury.

Priestly Smith recognizes that the Schiötz tonometer gives too low values, and believes the true normal intra-ocular tension to be generally about 24 mm. mercury. On this basis he estimates the average venous exit pressure should not be less than 25 mm. mercury.

Our observations show that the venous exit pressure oscillates above and below the intra-ocular tension. Accepting the value of normal

intra-ocular tension of 20 mm. mercury given by *Priestley Smith,* the venous exit pressure then would be about this value.

The blood-pressure in the veins, like that in the arteries, is subject to pathological modifications. We can infer that the venous exit pressure is below the intra-ocular tension when no pulse is induced by ocular compression. The difference, however, is usually but slight.

When the venous pressure is higher than the intra-ocular tension, its minimal and maximal values can be deduced from the degree of ocular compression required first to induce, and then to efface, the venous pulse. The difference between these values is quite small.

The appearance of a "doubled retinal pulse" occurs but rarely, and is apparently due to local vaso-dilatation. In these cases when ocular compression interrupts the venous current, it is probable that the venous pressure rises until the level of the arterial pressure is nearly reached. However, G. Lepiat believes that the venous pulse in these instances is but a reflection of the pulse of the arteries transmitted by the ocular mass.

The Granular Current.—Just after the arterial pulse has been overcome by ocular compression, and before the peripheral veins of the retina are effaced, the venous stream here breaks up into a mass of globules. Having,

then, no longer a "vis a tergo," the ocular compression squeezes the blood through the effaced central vein. The serum goes through first, the corpuscular masses follow. This "granular aspect" not only can be produced experimentally, but is also noted in certain pathological conditions.

Further Studies Relating to Ophthalmo-Dynamometry

In a recent paper, Priestly Smith[10] has analyzed in some detail the effects of external pressure on the eye. For the term "intra-ocular tension," he prefers "chamber-pressure" as being scientifically more precise.

The rise of chamber-pressure under a given weight, he finds, varies with the form and extent of the surface bearing on the eye: the smaller the surface, the greater the rise. A knob consequently raises the chamber-pressure more than a disc, as the latter affords a greater supporting area.

The chamber-pressure is less affected by an instrument when placed on the eyelid than when placed directly on the eye, and the smaller the terminal, the greater the difference. The interposed pad of the lid lessens the rise of pressure by enlarging the supporting area.

The rise of chamber-pressure under a given weight varies with the size of the eye; the

smaller the eye, the greater the rise. However, the errors likely to arise through difference in size of human eyes is generally negligible, except in young children and high myopes.

The rise of chamber-pressure under a given weight also varies with the character of the eye wall; the less distensible the tissues, the greater the rise. In children's eyes the tissues are definitely distensible, as continued access of chamber-pressure, we know, induces in them a uniform enlargement of the eyeball.

Equal increments in weight do not induce equal increment of chamber-pressure in a given eye. This, Priestley Smith notes, is visible in Bailliart's charts, and is to be expected as the distensibility of the eyeball diminishes as the chamber-pressure rises. Generally one finds, using Bailliart's dynamometer, a rise of 6.6 mm. mercury for each 10 gm. of force used.

In normal eyes, the venous exit-pressure and the chamber-pressure are usually nearly equal, and when either of them rises or falls the other does the same—a safeguard against edema and hemorrhage in the eye.

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CAPTER IV

THE CAPILLARY CIRCULATION OF THE RETINA

Recent studies indicate that the capillaries play a more active role than has hitherto been held. They do not simply dilate passively, more or less according to the amplitude of the arterial wave, but contract and expand independently in response to the chemical stimuli that are released in tissue metabolism. We are indebted to capillaroscopy for much new light on the physiology and pathology of the capillaries. Unfortunately, the retinal capillaries cannot be thus directly examined.

The Pressure in the Retinal Capillaries.—The blood pressure in the capillary bed is practically uniform. Its value cannot be directly measured, but we know it must be between the diastolic pressure of the central artery and the pressure in the central vein; that is, between 35 and 25 mm. mercury, or about 30 mm. Just as the pressure in the retinal arterioles becomes less the further the arteriole is from the main trunk, so the pressure in that portion of the capillary bed near the periphery is not so

great as in that portion near the disc. The value we have inferred as the average pressure in the retinal capillaries is similar to that which

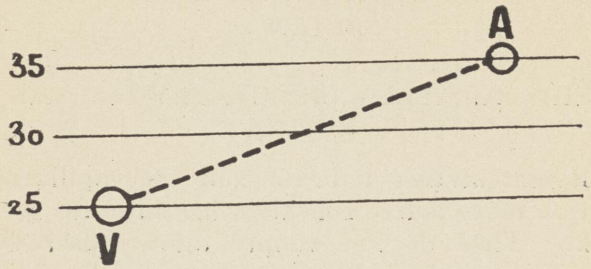

Fig. 9. Schematic figure, designed to represent the capillary pressure, which is necessarily intermediary between the arterial pressure, A, and the venous, V.

physiologists have found for the capillaries of the skin.

The Vasomotor Nerves of the Retina

Vasomotor phenomena, though occurring in the large arteries, have their principal sphere of activity among the arterioles. Vasomotor activity not only controls the amount of blood distributed to an organ, but interesting changes in pressure secondarily occur. With contraction of the arterioles, the systemic arterial pressure rises, and the systemic venous pressure falls. Reversely, when the arterioles are dilated, the systemic arterial pressure falls, and the venous pressure rises. The local circulation is affected quite otherwise. A constriction

of the central artery of the retina means a diminished output, and a fall of both arterial and venous pressure; while a dilatation of the artery means an increased output, and a rise of both arterial and venous pressure.

De Wecker[1] in 1889 made these statements in regard to the action of the cervical sympathetic on the eye: Electric irritation of these fibers increases the vascular tone, and dilates the pupil. With the ophthalmoscope one sees a perceptible diminution in caliber of both arteries and veins. These phenomena are very transient, as the irritability of the cervical sympathetic is rapidly exhausted.

Morat and Doyon[2] noted that the results of stimulation are different for different species; and may vary too in the same animal according to the segment of the sympathetic chain stimulated.

In the experiments of Magitot and myself[3] on the cat, excitation of the cervical sympathetic produced a contraction of the retinal vessels; and section thereof a dilatation. While measuring the retinal arterial pressure, we have observed it to diminish gradually as the vessels contracted under sympathetic excitation.

In man, Leriche[4] noted that after resection of the sympathetic sheath about the internal carotid, there was miosis, and dilatation of the retinal vessels on the operated side, thus prov-

ing that in man as well as in the lower animals the retinal arteries are supplied with vasomotor nerves. Aubaret[5] has directly observed these nerves about the central artery. Wölfflin[6] stimulated the cervical sympathetic in man with a resultant slight, but noticeable, contraction of the retinal arterioles, as well as those of the conjunctiva and uvea. Mydriasis, exophthalmos, widened palpebral fissure, increased lacrimation, and a rise in intra-ocular tension accompanied.

The vasomotor nerves of the retina reach the eye from the center of the medulla by way of the cervical sympathetic and the ophthalmic nerves, being associated in their course with the dilating fibers of the iris. Anterior to the Gasserian ganglion, the trigeminal nerve contains no vasomotor fibers.

The vasomotor nerves of the iris exert a much stronger action than those of the retina. Stimulation of the sympathetic momentarily blanches even large arterioles of the iris; but the retinal vessels are only just perceptibly affected. Only in exceptional cases do the retinal vasomotors reveal their presence clinically. They appear impervious alike to the effects of cold, heat, pain, and emotion. Their feeble action, assuring the smooth course of the retinal circulation, is undoubtedly a safeguard to these delicate tissues.

The Action of Drugs on the Retinal Circulation

Effects on the retinal circulation can be objectively measured by the appearance of the retinal vessels, and by local blood-pressure determinations; subjectively by changes in the visual acuity and the visual field.

The miotics have long been considered as vasoconstrictors; the mydriatics, as vasodilators (Abadie). Inspection, however, fails to reveal any change in vessel caliber resulting from the instillation of these drugs.

The inhalation of amyl nitrite, however, does produce some effect. Several seconds thereafter, the retinal venules and arterioles appear somewhat dilated, but only for a very short period.

Adrenalin, whether administered by instillation or by subconjunctival or subcutaneous injection, apparently leaves the retinal vessels unaffected.

Considered from the standpoint of local blood-pressure findings, somewhat more positive results are elicited. Atropine appears normally to produce a slight elevation of the venous pressure. For in many individuals in whom ocular compression failed to effect venous pulsation previous to the instillation of atropine, one-fourth hour thereafter venous pulsation could be thus induced. The arterial

pressure must hence be also slightly elevated, but this is seldom measurable.

Leplat[7] found that atropine distinctly increased the pressure in the iridic arteries, though without influencing the ocular tension. This is a noteworthy observation, as Magitot and I[8] have shown that the retinal circulation closely parallels that of the uvea.

Pilocarpine and eserine, according to Leplat, produce both a lowering of tension and a constriction of the iridic arterioles. It is possible that the retinal arterioles may be in slight degree similarly affected; but we, studying cases of aniridia and pupilary immobility with these drugs (as otherwise the miosis interferes with fundus observation), have been unable to note any appreciable pressure changes.

Instillations of cocain and of adrenalin likewise give negative findings as regards pressure changes.

The Subconjunctival Injection of Adrenalin.—After a subconjunctival injection of five drops of adrenalin (1:1000) there occurs a marked and prolonged diminution of ocular tension, and a lowering of the arterial and venous pressure. The appearance of the retinal vessels, however, remains unmodified. The systemic pressure was not appreciably affected by the amount injected.

The Subcutaneous Injection of Adrenalin.—Subcutaneous injections of adrenalin do not influence the ocular tension, even when the amount injected causes the systemic pressure to pass from 82/110 to 115/190. The pressure in the central artery of the retina rises with the increase of systemic pressure.

It appears then that in the subcutaneous injection of adrenalin the very finest arterioles are affected, whereas in subconjunctival administration the action is principally on the larger trunks (ciliary and retinal).

Amyl Nitrite.—A few seconds after the inhalation of amyl nitrite the systemic diastolic and systolic pressures for a brief period abruptly fall, to be followed in turn by a rise in pressure slightly above the original level. These phenomena are due to the opening and closing of the capillary bed.

During the action of the drug the ocular tension is definitely increased; but it still requires the same dynamometric pressures as previously used to induce the arterial pulse and then to cause its disappearance, thus indicating a rise in the diastolic and systolic pressures of the retinal arteries. This rise is in turn followed by a fall to slightly below the original value. The pressure changes in the central artery of the retina, as in the peripheral circulation elsewhere, are the reverse of those occurring in the large vessels.

Subjectively, the patient often notes, simultaneously with the throbbing in the head, an enlargement of the visual field. The visual acuity is unaffected.

Thus the retinal vasomotor mechanism is seen, all in all, to be but little influenced by drugs—a finding in harmony with the slight changes that ensue on stimulating the cervical sympathetic.

Subjective Phenomena Relating to the Retinal Circulation

Purkinje's phenomenon, which is so well known, was the first proof that the percipient layers of the retina must be posterior to the retinal vessels. The phenomenon is perhaps most easily elicited by taking off the head of an electric ophthalmoscope, and moving the lamp very gently over the closed lids. Soon the subject (who should be in a dark room) sees the retinal vessels projected before him—dark on a red background. The vascular net can be followed almost from the capillary extremities nearly to the disc.

The Capillary Circulation.—The capillary circulation is likewise subjectively visible, Says Giraud-Teulon[9]: "If one exposes the closed eyes for a few seconds before a strong light, such as that of a briliant white cloud, after several seconds one notes the red field crowded with a number of contiguous eddies of

about equal diameter. Little globules dance around in the circumference of each eddy. The appearance is reminiscent of the microscopic

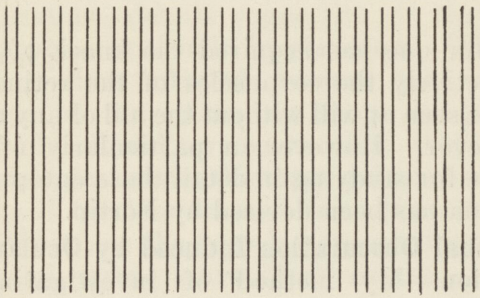

Fig. 10.

view of the interdigital membrane of the frog; in truth, revealed before us is the spectacle of the retinal circulation."

This phenomenon can be seen, too, by regarding directly a white cloud, or looking at the cloud through a colored glass, preferably yellow. One will probably, however, observe it best by carrying out the following procedure: On a white card, 50 cm. square, draw lines with ink one-half cm. apart. The card should be in good light, though not directly illuminated. Gaze then at the lines without trying to focus them clearly. When the lines are held vertical one sees between the card and the eye a horizontal movement of thin points, moving both

from left to right and vice versa. With the lines horizontally, the movement appears vertical—like a rain of minute globules. When the head is tilted, the rain takes an oblique direction.

After one has acquired the knack of seeing subjectively the corpuscles of the retina, they can be seen at will without the aid of any artifice whatever. However, a particularly effective device for studying in detail these entoptic phenomena has been devised by Fortin.[12]

Visual Obscuration Induced by Ocular Compression.—My method of investigating the visual obscuration produced by ocular compression has been this: The subject closes one eye and fixes a light with the other. As this fixing eye is compressed, complete suppression of vision ensues, the nasal field disappearing first. Just before obscuration takes place, the subject may perceive against the hazy background the rhythmic pulsation of a filiform shadow, or of a dot rhythmically enlarging and contracting. These pulsatile sensations have also been noted by Reich.[13]

These phenomena are thus explained: The circulatory arrest induced by compression is forthwith followed by arrest of retinal function. A similar instantaneous loss of function occurs in the brain when any of its circulatory supply is obstructed—another point of paral-

lelism between these delicate tissues.

The visual suppression induced by compression proceeds from the nasal to the temporal field, and is preceded by a period of relative obscuration—details suggestive of that which takes place gradually in glaucoma. The nasal field is first affected because the temporal vessels of the retina are more readily compressed. Rydel[11] remarks that even in the physiological state, the temporal portion of the retina is the less favored from the circulatory standpoint, as the vessels here are fewer, thinner, and pursue a less direct course.

The subjective sensation of pulsation corresponds to increased pulsatile excursions of the artery, and can be plausibly explained as the shadow cast on the visual cells by this arterial movement.

Suppression of vision does not appear until the intra-ocular tension, as affected by compression, becomes at least equal to the systolic pressure in the tributaries of the central artery. When the intra-ocular tension becomes equal to the diastolic pressure, pulsation may be perceived.

In the normal subject, 10-15 seconds will lapse after the necessary pressure is exerted before total suppression of vision ensues. In patients with hypertension, this interval is prolonged and may reach a minute or more. In

patients with hypotension, the interval is considerably reduced. The passage of blood in the retinal arteries varies under these different conditions. When the intraocular tension equals the systolic pressure, no new blood can enter the retinal arteries; but the blood already in these vessels continues to flow toward the capillaries, propelled by the ocular compression. The less the peripheral resistance the faster the blood is forced through, and the quicker suppression of vision occurs—and vice versa.

The interval required to induce suppression of vision is the same whether the individual fixes on a white or a colored light. The compression produces, however, interesting changes in color perception. Before suppression occurs, red disappears and is replaced by a brilliant white. If both eyes fix, and compression is practiced simultaneously on them, with the induction of blindness, the visual field is covered with squares of brilliant yellow.

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CHAPTER V

THE DISORDERS OF RETINAL CIRCULATION

Functional Disorders

In an eye of normal tension, if the induction of the arterial pulse requires more than 30 gm. dynamometer pressure there is diastolic hypertension, i. e., more than 40 mm. mercury; and if the suppression of the pulse requires more than 80 gm. dynamometer pressure, there is systolic hypertension, i. e., more than 80 mm. mercury. One can diagnose pronounced cases of retinal hypertension without apparatus by noting the stronger finger pressure required to suppress the arterial pulse. Though it is more difficult, the practiced finger may even recognize, also, when it requires more than usual pressure to induce the arterial pulse.

Measured by the dynamometer, the pressure in the central artery of the retina may reach to 120 mm. diastolic. The heights that its systolic pressure may reach we cannot at present measure, since it is both impractical and inadvisable

to exert with the dynamometer a pressure greater than 150 gm. When this pressure does not suppress the pulse, we merely note: Retinal systolic pressure > 150 gm.

The Diagnosis of Retinal Hypertension.—Among the principal signs of retinal hypertension, according to the usual view, are anomalies in caliber and tortuosity of the vessels and miliary aneurisms[1]; but these findings are inconstant. Other features described[2] are: Hyperemia of the disc and distention of the vessels; widening and increased brilliance of the reflex band on the arteries; denting of the veins by overlying arteries; and pallor and narrowing of the arteries. Adams[3] considers this last point particularly valuable, and places no reliance whatever on variations in the reflex streak.

On the basis of personal experience, however, I am perfectly convinced that hypertension may exist in the retinal vessels without any noticeable alteration in their appearance. The sole objective sign is often that furnished by the dynamometric study of the reactions of the arterial pulse to ocular compression.

Sudden attacks of visual obscuration occurring in apparently healthy subjects is suggestive of retinal hypertension. Black specks that come and go independently of the ocular movements is a suspicious complaint that is perhaps

most frequently elicited. An analysis of the character and movement of these specks by the more intelligent patients tends to indicate that we are here dealing with an exaggerated subjective sensation of the retinal circulation.

Occasionally there is noted in the periphery of the visual field, instead of black specks, a movement of sparkling points that abruptly appear and disappear. A normal subject will sometimes experience this sensation after some sudden intense strain, such as may occur in violent sneezing. This phenomenon, quite contrary to that of "phosgene," is only seen in the light, and with the eyes open. Though difficult to explain, its presence is generally associated with retinal hypertension.

Many patients with vague visual disorders often yield completely negative objective findings. Though these individuals complain of seeing objects through a haze, the visual acuity and the visual fields may be normal. This subjective condition is usually ascribed to a failure of accommodation, and glasses are accordingly ordered; but in reality these are often cases of retinal hypertension. It is important that such a condition be not overlooked, for, if warned by the local increase of tension, we locate the cause and treat the patient accordingly, we may avert further inroads and generalized changes.

The finding of retinal hypertension may be

the first suggestion of systemic involvement. Leplat[4] cites a case in point: A laborer, age 32, a heavy eater and well developed, complained of a haze constantly disturbing his vision. The ophthalmoscopic findings were negative, but the blood-pressure in the central artery was: Diastolic, 70; systolic, 150. The systemic blood-pressure was: Diastolic, 170; systolic, 250. The Wassermann test being negative, general measures were instituted, including regulation of diet and purgation; Potassium iodide was prescribed. In two months the haze had completely disappeared; the retinal blood-pressure had gone down to: Diastolic, 60; systolic, 120; the systemic pressure was down to 130/205. The patient then discarded his regimen and resumed his former habits, with a recurrence of all symptoms resulting a month later.

I have had similar cases.[5] It is important, then, not to depreciate the patient's subjective symptoms of flitting specks, haze, and occasional obscuration. The basis of these sensations is, in some cases, probably arterial spasm; in others, perhaps incipient edema of the retinal tissues—too slight to be perceptible with the ophthalmoscope.

The Causes of Retinal Hypertension.—Hypertension of the retinal vessels is most usually associated with systemic hypertension.

Normally, the retinal diastolic pressure is .45 of the systemic diastolic pressure; but in hypertension cases the retinal diastolic pressure is more than proportionally elevated. In three patients, for instance, with respective systemic diastolic pressures of 120, 140, and 160, the retinal diastolic pressure, according to my records, was, respectively, 60, 80, and 86 mm. mercury. The retinal diastolic pressure is particularly high in the retinitis of pregnancy and in albuminuric retinitis.

Because the retinal systolic pressure cannot very well be measured above 150 mm., we have been unable to determine how the retinal systolic pressure is affected in these cases.

Local vasometer phenomena are the most important factors in causing the retinal blood-pressure to vary from its usual relations to the systemic pressure.

The Diagnostic Value of Retinal Hypertension.—A brief consideration of the various conditions in which retinal hypertension is found is now in order:

1. Retinal Hypertension Accompanying Systemic Hypertension.—Whenever the determination of the retinal blood-pressure shows an abnormally high value, we should first consider systemic conditions as the probable basis.

Retinal hypertension reaches the highest peaks in albuminuric retinitis, and in the ret-

initis of pregnancy. An important point in these cases is that the local hypertension may precede all other ophthalmologic symptoms.

In diabetic retinitis, and in the hemorrhagic retinitis of the aged, the hypertension is much less pronounced, oftentimes the retinal systolic pressure alone being elevated.

In obscure forms of retinal hypertension syphilis is to be thought of as a frequent factor.

2. Retinal Hypertension Unaccompanied by Systemic Hypertension.—In optic neuritis, before the stage of atrophy, a low degree of retinal hypertension is present.

With increased pressure in the cerebrospinal fluid, the retinal blood-pressure is frequently elevated, often without the systemic pressure being changed. Bollock and Mengot de Treigny[6] noted in a child who manifested ocular signs after skull fracture a retinal hypertension with a normal systemic pressure. Lumbar puncture demonstrated that the cerebrospinal was under tension. A week later the pressures in the cerebrospinal fluid and the retinal arteries had returned to their normal values. Two other cases of skull fractures, with similar findings, have been personally observed.

A very interesting case was observed by Morax and Lagrange[11]. A woman, aged 34, complained of sporadic sensations of visual fog. Examination revealed bilateral choked disc, and

a moderate degree of retinal hypertension. Lumbar puncture was made and a small amount of fluid removed. Seventeen hours later the patient became hemiplegic and comatose, and died the day following. Autopsy demonstrated a tumor in the aqueduct Sylvius and a large sub-dural hemorrhage. Due to the hypertension of the cerebral vessels, the relatively slight decompression had sufficed to induce a fatal hemorrhage.

How is this relationship between the pressures in the cerebrospinal fluid and in the retinal arteries to be explained? The circulation in the retina generally reflects that of the brain, and when retinal hypertension occurs we can usually infer cerebral hypertension. Intracranial pressure may cause a rise, too, in systemic pressure. The rise, whether local or general, is an effort to compensate for the anemia that would be otherwise produced. A relationship probably also exists between the systemic blood-pressure and that of the cerebrospinal fluid, for lumbar puncture is capable of lowering the former when excessive[7].

In pregnancy, retinal hypertension may occur without any change in the ophthalmoscopic picture or in the systemic pressure. This localized hypertension appears to explain the retinal hemorrhage in the following case: A primipara, 39 years old, whose general health had been

good, noted suddenly a black spot before the left eye. The ophthalmoscope revealed a single pre-retinal hemorrhage of about one disc diameter in the macular region; there were no exudates. The right eye was unaffected; the intra-ocular tension of each eye was 20 mm.; and the pressure of the central artery of each eye, 70/120. The systemic pressure was: 80/125. There was no albuminuria and the blood area was normal. The ophthalmoscopic appearance a week later was the same, but the retinal blood-pressure was nearer normal: 45/95 mm. mercury.

Complications of Retinal Hypertension

The most formidable danger of retinal hypertension is hemorrhage. Though retinal hemorrhage rarely comes from the arteries, still hypertension in the arteries means hypertension also in the capillaries and veins; and these vessels support increased pressure with difficulty. Hemorrhage may occur without lesions of the vessel walls, but prolonged hypertension deleteriously affects their nutrition with resultant plaques of atheromatous degeneration. Over-development of the muscular coat, engendered as a compensating effort, is the basis of angiospasms, to be considered in a special detail later.

Hypotension of the Retinal Arteries

Retinal hypotension is less frequently encoun-

tered than retinal hypertension; it generally accompanies a diminution of systemic pressure, and is most clearly marked in syncope. Just before the latter occurs a spontaneous arterial pulse is observable.

DeWecker[8] has graphically described the ocular phases of hypotension: "As the subject falls into a swoon, the vision becomes instantaneously dim; the weakened cardiac action producing a depression in both the cerebral and retinal circulations. Similarly, at the time of death, as the circulation gradually fails, the dying man is plunged into the shadows while perhaps still maintaining consciousness. The same darkness engulfs the unfortunate epileptic as the first warning of an attack."

In aortic insufficiency, the diastolic pressure is extremely low, so that the arterial pulse is either present or induced by minimal pressure. Nunes[9] reported a case where this observation revealed a hitherto unsuspected aortic leak. The patient's diastolic pressure alone was affected, both the systemic and retinal systolic pressures being normal.

Local hypotension is associated with the various degrees of obliteration of the central artery of the retina. The most striking instances are found in the different types of optic atrophy. Though the systolic pressure is lowered, the diastolic is practically unaffected.

In a case of acromegaly under my observation, there was a local hypotension of the retinal arteries with a normal systemic pressure.

In pulsating exophthalmes[12], localized hypotension exists not only in the proptosed eye, but in its companion as well—demonstrating how a deficiency in one carotid will affect the entire cerebral circulation. In such cases, the retinal systolic pressure may be only slightly above the diastolic.

In chorioiditis, whatever its nature, local hypotension generally occurs.

Hartmann[13] has observed that after retrogasserian neurotomy there is a definite and constant lowering of the retinal arterial pressure.

The Consequences of Retinal Hypotension.—Retinal hypotension means insufficiency of the retinal circulation. This condition occurs acutely in syncope, severe hemorrhage, and before death. The cases just considered illustrate how it may persist in a state more or less chronic. Disturbances of the retinal circulation may be due to purely local causes, but most often are the reflection of cerebral conditions.

In a rare case of obliterating arteritis of the right carotid, DeMeyer[10] noted a vertiginous state, a tendency to faintness, with slight disturbances in sight and hearing, but subject to rapid fatigue of these senses. In a patient I observed, in whom both common carotids had

been successively tied, the findings were similar. He could not read for more then ten minutes without having a sensation of dazzling. Stooping affected vision and induced vertigo. His vision was: R. 20/80, L. 20/60; his retinal blood-pressure: R. 32/40, L. 30/45; his systemic pressure: 95/130. His fields were concentrically contracted. A consulting otologist noted a hypo-excitability of the labyrinths. This suggests that aurists may find the investigation of retinal blood-pressure of some importance in patients presenting labyrinthine symptoms.

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CHAPTER VI

DISORDERS OF THE VENOUS CIRCULATION

A venous hypertension exists as well as an arterial hypertension[1]. The presence of a spontaneous venous pulse, however, means nothing in this regard, as this phenomenon is noted as often in hypotension as in hypertension cases. The evaluation of venous pressure depends somewhat on the conditions encountered. The following cases are to be considered:

1. Where a Spontaneous Venous Pulse Exists.—When a venous pulse is present, the venous pressure must be in equilibrium with the intraocular tension, being, by a few millimeters, above the latter in diastole, and below in systole. The slightest compression of the eyeball generally extinguishes this spontaneous pulse. But if the venous pressure is above normal, correspondingly more compression must be exercised. When, then, the venous pulse persist after a dynamometer pressure of 25 mm., venous hypertension exists and the value of its maximal level is indicated by the dyna-

mometer pressure necessary to cause its disappearance.

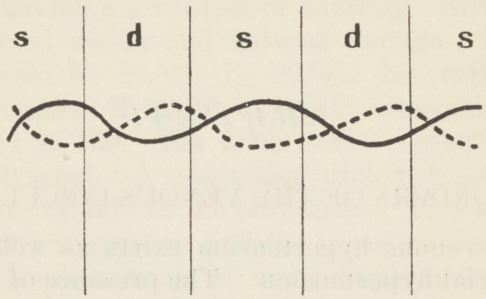

Fig. 11. Schematic representation of spontaneous venous pulse. The venous pressure (dotted curve) is lower than the ocular tension (line curve) during systole, and exceeds it during diastole. The vein collapses at the moment when the venous pressure is below the ocular tension.

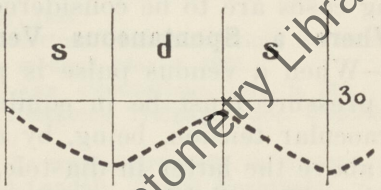

Fig. 12. The ocular tension has been raised again to 30 mm. by compression of the globe; the venous pressure being constantly below the ocular, there is no venous pulse.

2. Where a Spontaneous Venous-Pulse Does Not Exist.—If the venous pulse does not exist spontaneously and its appearance is induced by ocular compression, the venous pressure must

be higher than the intra-ocular tension originally present. The pulse appears when the intra-ocular tension, as modified by the outside pres-

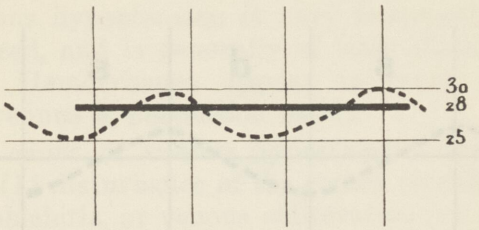

Fig. 13. The ocular tension has again been raised to 28 mm. The venous pulse persists, because during diastole the venous pressure (dotted curve) remains higher than the ocular.

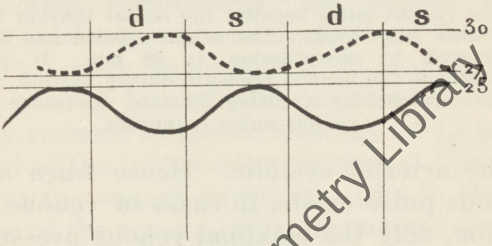

Fig. 14. There is no spontaneous venous pulse, because the venous pressure is continually, even during systole, higher than the ocular tension.

sure, is just above the minimal venous pressure, but below its maximum. The dynamometer reading at the appearance of the venous pulse indicates the minimal venous pressure in these cases; but, owing to secondary changes, further ocular compression will not suppress the

pulse at the point of maximal venous pressure. In the obstructed venous circulation, the back pressure rapidly mounts until it becomes equal

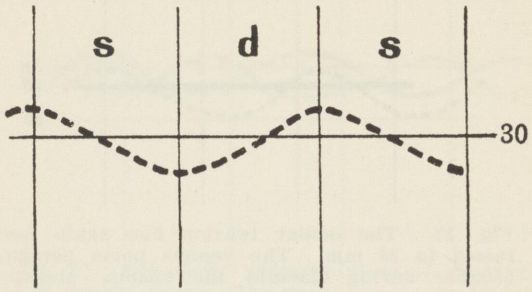

Fig. 15. The same case as Fig. 14. There was no venous pulse because the ocular tension was below the venous. The ocular tension has been raised, by compression, to 30 mm. It now exceeds the venous pressure during systole, and is exceeded by it during diastole; therefore the venous pulse reappears.

to the arterial systolic. Hence when a spontaneous pulse exists, in cases of venous hypertension, only the maximal venous pressure can be measured; when it does not exist, only the minimal pressure can be reliably determined.

At times it is particularly easy to recognize venous hypertension. Should the maximal venous pressure be greater than the minimal arterial pressure the venous pulse will still persist, after the dynamometric pressure has induced the arterial pulsation. When this "doubled retinal pulse" occurs (the arterial

pulse marking systole, the venous pulse, presystole), there is a definite pathology in the return circulation.

Venous hypertension is very frequently encountered, and is generally of little diagnostic value. Hemorrhages appear to occur more when venous hypertension exists.

The cause of venous hypertension may be either (1) disturbance of the return circulation, as in phlebitis, or venous obliteration; or (2) a peripheral vaso-dilatation, which more fully exposes the capillaries and veins to the arterial pressure.

In *venous hypotension,* the venous pulse neither exists spontaneously nor can it be induced by ocular compression. It may appear, however, when the intra-ocular tension is sufficiently reduced by gentle massage. The measurement of the intra-ocular tension at this time will give the venous pressure. Due to the resistance of the venous walls, the retinal circulation can continue with an intra-ocular tension greater—though not much greater—than the venous pressure. The lowest venous pressure occurs in chorio-retinitis, for in this affection the power of the blood stream becomes reduced by a capillary obliteration of greater or less extent.

The Pathological Granular Current.—A granular current, similar to that produced in the

larger veins by prolonged ocular compression, is seen often after the sudden obliteration of the central artery. It sometimes occurs in both arteries and veins, but generally the venous column alone is affected. The broken column is, however, immobile, unless the circulation becomes re-established.

The granular current is occasionally seen in advanced cases of luetic chorioiditis, in which there is more or less obliteration of the capillary bed. As the force of the arterial stream is no longer transmitted, the expression of the venous blood becomes dependent on the intraocular pressure. Ploman[2] has discussed this subject in some detail.

The Color of the Blood in the Retinal Vessels.—DeJaeger[3] and Giraud-Teulon[4] have tried to correlate differences in color between the arterial and venous blood with changes in the circulatory function of the retina; the less marked the difference, the less active the changes in the capillary bed, and vice-versa. Henocque[5] has tried to study the retinal vessels spectroscopically.

In the lipemia retinalis of diabetics, first noted by Heyl, the retinal vessels have a distinctive aspect. The vessels—especially those in the periphery—are almost white on the red background, and it is no longer possible to distinguish by color arteries from veins. This ab-

normal appearance is due to the high lipoid content of the blood (25 per cent).[6 7]

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CAPTER VII

RETINAL ANGIO-SPASMS

It has long been known that attacks of total or partial obscuration may affect one or both eyes suddenly for a time, and then pass, without any noticeable ophthalmoscopic lesion being present, except perhaps pallor of the disc, and more or less contraction of the vessels.

Vascular disorders of this type are quite frequent. Those having a cortical origin, such as ophthalmic migraine, must be distinguished from those affecting the retinal vessels. In the former group, the obscuration often assumes the form of hemianopsia, or may be total, and the blindness is generally absolute. The patient does not have even light perception in the half or the whole of the field affected. Cases of this type have been reported in hypertension[1], plumbism[2], and acute nephritis[3]. The diagnosis of ophthalmic migraine presents no difficulty. In these blind attacks of cerebral origin, scintillating scotomata often precede, accompany, or follow the crises.

The obscuration due to spasm of the retinal vessels has not been so extensively studied.

Here the visual loss is generally relative, rather than absolute. The attack may involve one eye or both; the entire visual field or a sector thereof. A haze more or less dense comes on suddenly, disturbing enough to interfere with reading and close work, but permitting the patient to go about without help. At the worst, hand movements are visible. Occasionally a mild frontal or orbital headache accompanies the visual disturbance, or there may be a "tightening sensation" behind the eye. A slight dilatation of the pupil generally persists for a while, with a certain degree of paralysis of accommodation.

The obscuration is most often limited to one eye, while in cortical angiospasms, both eyes are always affected. Hemianopsia occurs but exceptionally. When only a branch artery of the retina is affected the obscuration is limited to a sector of the visual field.

The Ophthalmoscopic Findings in Retinal Angio-Spasm.—As the patient generally visits the oculist one or more days after the attack, the opportunities of noting the early ophthalmoscopic changes have been limited. The cases fall into two clinical groups:

1. The spasm affects the trunk of the central artery of the retina, or all its branches.—Dickson Bruns[4] observed these details in a young woman who complained of sudden visual

disturbance: the arteries were narrowed, the veins dilated and dark, and there was a bluish halo about the disc (edema). Vaquez[5] cites a case of similar type in which the disc pallor yielded to an inhalation of amyl nitrite. In a patient of Weiss'[6], the vessels were contracted to white lines, but in a half hour later were refilled.

Bruner[7] had under observation a young man of 34 who was subject to recurrent attacks of transient blindness. The vision would diminish to the point where hand movements were no longer perceptible. The retina became pale, the circulation arrested, and the blood column in the veins fragmented. Then, after one to three minues, pulsation appeared, the arteries recovered their caliber, and vision very rapidly came back. In this case there was an extreme degree of arterial obliteration. Such cases are easily confused with thrombosis or embolus of the central artery. Differentiation depends on the history of the patient and the suddenness of the appearance and disappearance of the attack. Unless changes are taking place at the time of the examination, the ophthalmoscope does not give much aid; but occasionally while looking at the vessels, one sees them suddenly refill. [Another important point is this: in spasm, ocular compression may still induce an arterial pulse; not, however, in thrombus or

embolus.] After the attack the appearance of the fundus is entirely negative.

2. Spasms of a branch of the central artery. —The arterial spasm, after affecting the central artery, often localizes in a single branch.

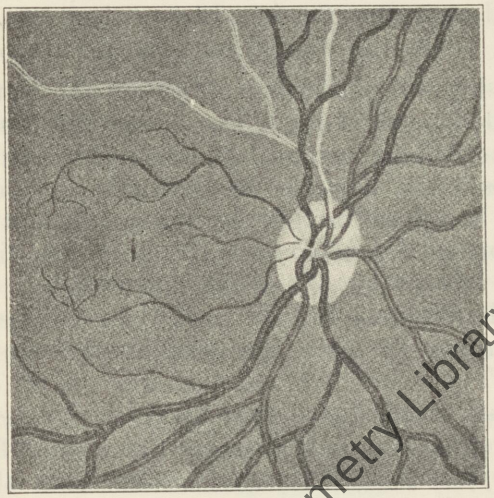

Fig. 16. Retinal spasm. (Koby.)

The subjective disturbance remains particularly marked when a macular artery is affected.

The ophthalmoscopic signs persist as long as the visual trouble—usually for several days. The caliber of the involved vessel is hardly diminished, but from one end to the other of its course it is ensheathed in a distinct white track. The following case is characteristic:

A man of 34 years suddenly noted shortly after awakening a visual disturbance, which, slight at first, developed into a relative blindness that gradually extended to the totality of both visual fields. During this attack the patient could go about his room, but was unable to read or write, nor dared he venture out. In twelve hours the disturbance passed away.

I had the opportunity of seeing the patient the following morning. The vision of the right eye was still somewhat diminished; the left eye was absolutely normal. In the right eye the superior temporal artery lay in a white track and appeared somewhat contracted. Its visual acuity was 20/30. The field was definitely contracted infranasally. In both eyes the ocular tension was 22 mm.; the local blood pressure, 32/70. The systemic blood pressure was 90/135; the heart and kidneys were negative. Valerian was prescribed. A week later the vision of the right eye was 20/20, and the white zone about the superior temporal artery had almost disappeared.

Koby[8] has observed a case that is even more striking. The left visual field showed a defect almost exactly below the horizontal line. The superior arteries were extraordinarily thinned —some branches were invisible, and others showed but a filament surrounded by a white sheath. The fundus picture would suggest that

the arterial spasm was superimposed on a chronic obliterating arteritis.

Ophthalmoscopic signs are not always present. A patient with a sector defect in his field of vision visited me after having already consulted various oculists who found the fundus entirely normal. While the local blood pressure was being determined from the pressure of the dynamometer he suddenly became well.

The Effects of Arterial Spasm Upon the Retina.—The degree that arterial spasm affects the retina depends on whether the vessel involved is totally or partially closed, and on the duration of the spasm. In Bruner's case, though the circulation was completely arrested, the spasms lasted but one to three minutes, and the patient's vision completely recovered each time.

The attack is usually very brief, but may vary from a few minutes to a few hours in extent. The obscuration sometimes persists in attenuated form for several days. Generally there is but one attack, but occasionally the attacks recur from day to day, as in the following case:

A woman, now 22, had at the age of 18 a severe attack of articular rheumatism complicated by chorea. Since then she has had some visual disturbance almost daily. The first attack came during her illness, when she tried to

arise from bed. For about fifteen minutes she then suffered an almost absolute loss of vision. The attacks succeeding have been gradually lessening in severity. The vision is: R. 20/20; L. 20/30. The local and systemic blood pressure are normal, as are also the heart and kidneys.

In a case such as that cited, where the patient complains of an intermittent disturbance of vision, the diagnosis may be quite difficult. The ophthalmologist must be absolutely certain that no fundus lesion exists, except what may be incident to vascular spasm. After an attack of angio-spasm the visual acuity and the visual field are as before, even though there persists, as frequently happens, a sensation of slight haze. Should this haze immediately disappear after the inhalation of amyl nitrite the diagnosis is assured.

The ophthalmoscopic picture at the height of an attack may simulate that seen in embolus of the artery. However, the transitory character of angiospasm differentiates these conditions. But during the attack, even with the red macular spot in view, they can still be distinguished, for if the artery involved is affected by spasm an induced arterial pulse can still be elicited, but *not* if the vessel is actually obliterated.

It is unusual for vascular spasms to affect only the retina; generally there is a history of

analogous disorders in other parts of the body, as numb fingers, vasomotor disturbances, scintillating scotomata, speech defects, aprosexia, or—rarely—transitory hemiplegia. In a case of retinal angiospasm, seen after the ophthalmoscopic evidences had passed, a history of such associated phenomena previously would help suggest the diagnosis.

The Etiology of Retinal Angiospasm.—Vaquez ascribes cortical angiospasms to paroxysmal hypertension; the cause for retinal angiospasm is probably similar. But how does hypertension induce angiospasm? Does the vessel close to defend itself against an exaggerated pressure that menaces its walls? Or is it sequential to an overdevelopment of arterial musculature that the hypertension has induced? But let us consider the principal hypothesis.

Patients, ill with nephritis or plumbism, and already affected with retinal lesions, often experience a transitory aggravation of their visual troubles. An added haze makes their vision still poorer. These attacks may very well be explained by angiospasm on an hypertension basis. But hypertension cannot be the sole factor; in over half my patients the arterial pressure was not above normal. It has been advanced that these cases are subject but to occasional periods of hypertension—but why,

then, this transitory hypertension? This is said to be due to an exaggerated sensitivity of the sympathetic nerves, but what causes this hypertonicity?

Raynaud had observed spasm of the retinal vessels among patients afflicted with the disease that bears his name. The following is a very definite case that came under my observation:

A woman of 23 had had three years earlier a stormy pregnancy with albuminuria, convulsive seizures, and intermittent fogginess of vision several times daily. After term the ocular disturbance persisted, becoming more aggravated with time, so that the patient found herself unable to work for several hours each day. Between attacks the vision was normal. Vaquez, who studied the case, found the heart normal, Wassermann negative, no albuminuria, normal blood area, blood pressure 100/160. The hands became sporadically blue and cold. The retinal blood pressure, taken between attacks, was 45/100.

Considering that toxins have undoubtedly a disturbing action on the sympathetic, Grasset, in 1905, attributed arterial spasms to deficiency in the antitoxic mechanism. It is true that such probably occurs in nephritis, arteriosclerosis, and renal insufficiency, but most of our patients gave only negative evidence of intoxication—no albuminuria, no abnormal blood area, no

chronic infection. Apparently healthy young subjects, mostly women, are those principally affected. It must be admitted that there is much yet to be learned about the etiology of angiospasm.

The endocrines may sometimes be at fault. I observed a case[8] that was very definitely one of thyroid insufficiency. In several attacks of obscuration she was each time relieved by amyl nitrite.

Chronic glaucoma, it should be noted, is occasionally exaggerated by intervening retinal angiospasms, as the following case illustrates:

A watchmaker, age 62, noted on awakening two days before the consultation that the left eye was almost blind. This sensation disappeared in 5-10 minutes, to recur later that day and the day following. "A tightening sensation at the back of the eye" accompanied these attacks. The ophthalmoscope disclosed a marked excavation of both discs. The ocular tension was: R. 26 mm.; L. 38 mm. The retinal blood pressure was R. 35/65, L. 40/63; the systemic blood pressure was 90/160.

Spasm of the central artery is, too, very frequently a premonitory symptom of thrombosis of the vessel.

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CHAPTER VIII

ABNORMALITIES OF THE RETINAL VESSELS

The lesions of the retinal vessels will be principally considered from the ophthalmoscopic and clinical standpoints, with but incidental attention to details of microscopic pathology.

Anomalies

The divers ways in which the central vessels of the retina divide and subdivide have already been mentioned. So varied are the forms that their study should be of some help in anthropometric identifications.

Not infrequently an arterial loop springs from the disc, arising generally from the central artery and penetrating the vitreous. This condition, unusual in man, is constant in certain vertebrates, as in the roach fish. The anomaly must be considered congenital, and is seen in eyes otherwise normal[2] [3]. As is well shown in the adjoining sketches (Fig. 17, 18), a moderate sized arterial branch penetrates the vitreous in the form of a loop, the two parts of which are twisted on each other. In Kipp's case[4] the loop

penetrated 2 mm. into the vitreous, twising four times about itself, and returned then to the disc to proceed on its usual course. Ocular compression induced pulsation of the loop.

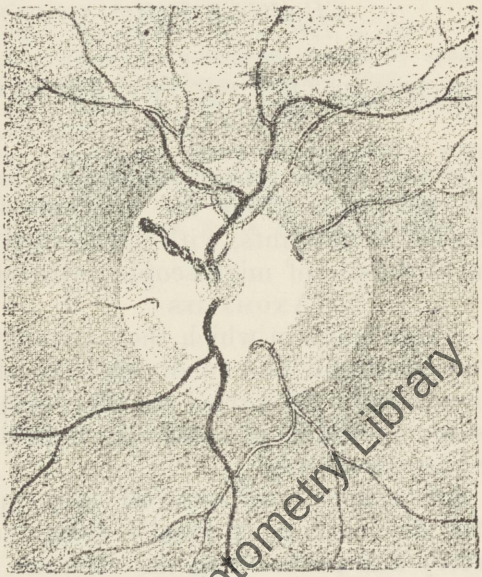

Fig. 17. Arterial spur projecting into the vitreous. (Ginsberg.)

Remnants, more or less complete, of the hyaloid vessels likewise penetrate the vitreous, but unlike the anomally just considered, the arterial stem does not return to resume its course in the retina. In fetal life the hyaloid artery goes for-

ward from the disc to end in a network on the posterior surface of the lens. It passes away shortly after the disappearance of the pupillary membrane. In examining the fundi of fifteen infants (new-born or but a few months old) Terrien[5] noted constantly that a slight vestige

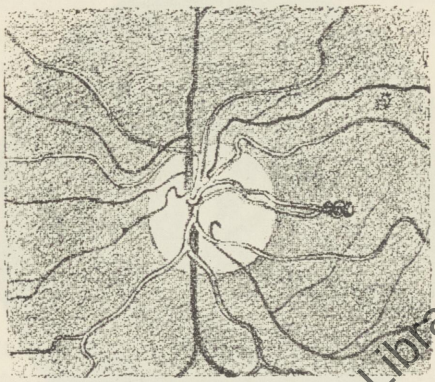

Fig. 18. Retinal arterial spur projecting into the vitreous. (C. Hirsch.)

of the hyaloid artery still persisted in the form of a tiny filament, 1-1½ mm. in length, arising from the nasal portion of the disc and extending into the vitreous.

Occasionally, as in cases reported by Danis[6] and Ruhlwandl[7] a considerable part of the hyaloid artery persists to adult life. In these cases the vestige existed as a distinct cord from which arose fine extensions (vessels of the vitreous), and on the posterior surface of the

lens was a filamentous network. The vestige sometimes ends, however, in knob-like terminations (DeBeck[8]).

Vassaux[9] reports that an eye enucleated by Panas for supposed glioma proved to be, in-

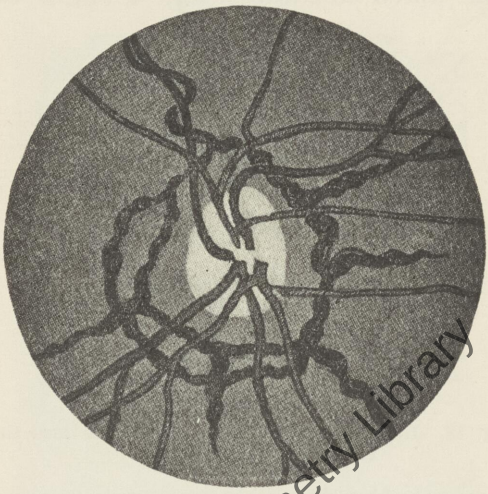

Fig. 19. Abnormal peri-papillary vascular ring.

stead, a cystic degeneration of the anterior network of the hyaloid artery. Dor[10] believes that perivasculities of persistent hyaloid vessels may account for certain obscure affections of the lens.

A very curious anomaly is that observed by Coppez[11] and named by him "the peripapillary

vascular ring'' (Fig. 19). The case cited concerned a woman of 39, who, without any ocular disturbance, presented about each disc a highly developed venous plexus.

Cases have been reported in which the course of the vessels was so sinuous as to suggest the turns of a helix (Levin[12], Dor[13], Ducamp[14]). This helicoidal state, however, represents but a congenital anomaly, and is without diagnostic or prognostic significance.

Landolt has noted that the vessels in hypermetropic eyes tend to be rather tortuous. Owing to the relatively smaller size of the eye, the vessels have a lesser area to cover, which probably accounts for their serpentine behavior.

Localized Dilatations of the Retinal Vessels

Retinal Varices.—Varices of the retinal veins are exceedingly rare, probably because, in contrast to the vessels of the lower extremity, the retinal vessels are protected from special strains.

Dilated and tortuous vessels appearing after thrombosis of the central vessels do not represent true varices, but rather a varicose condition. Marc Landolt[15] reports an interesting case in which he makes this distinction. The fundus (Fig. 20) showed a very thin communication between two primary branches of the central artery. Nearby, arising from a primary

branch of the central vein, was a corkscrew venous cul de sac. Landolt assumes that the condition followed a circulatory obstruction occurring somewhere in the optic nerve between

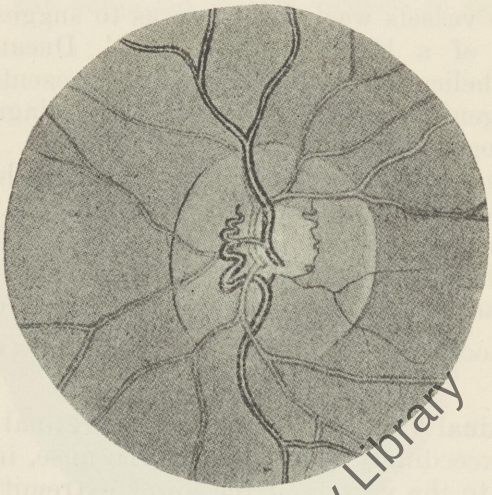

Fig. 20. Varicosities of the disc. (Coppez.)

the disc and the point of reunion of the two branches of the vein.

Liebreich[16] and Schiebl[17] have reported very unusual cases in which the retinal veins have a bead-roll appearance, due to the presence of successive small dilatations (Fig. 21):

Arterial Aneurisms.—Very rarely does aneurism affect the retinal arteries. When present, they are generally seen on or adjoining the disc.

The dilatation is usually very small, pulsates when the artery pulsates, and the color thereof is somewhat lighter than that of the unaffected artery. These aneurisms are mostly secondary to arterial obliteration; more rarely primary[18, 19]. Sometimes there is shown an aneurism on the disc, accompanied by an obliteration of the inferior temporal artery. In this case it is easily understood that the arterial wall dilated to the increased pressure occasioned by the obstruction.

In primary aneurism[20, 21, 22] a lesion of the arterial wall exists, most often luetic.

Von Hippel's Disease.—Von Hippel described in 1903 a distinctive affection of the retina, and since then there has been about 20 to 30 cases reported[23].

The characteristic feature in the fundus is the appearance of red ball-like formations, projecting more or less from the retina, and attaining sometimes the size of two disc diameters, though generally smaller. They occur in variable numbers and are distributed indifferently in any portion of the field. Ocular compression causes their effacement. These formations are always intercalated between arterial and venous branches. The vessels take a sinuous course in entering or leaving these little globes, and the color of the arterioles and venules associ-

ated with them is so similar as to make their differentiation difficult.

In advanced stages of the affection, white spots appear near the formations, and often

Fig. 21. Moniliform retinal veins. (After Schoebl.)

retinal hemorrhages. Secondary changes produce the picture of retinitis proliferans; detachment of the retina may occur, and changes in the vitreous. The prognosis is bad, blindness gradually supervening. In the progress of the disease the ocular tension may be increased or decreased.

Histological studies in this condition have

shown, besides vascular changes, an abnormal proliferation of glial tissue (VanDuyse[24], Arganaraz[25]). The etiology is unknown. There appears to be a congenital basis, though not till the third decade do symptoms appear. Several members of the same family have been affected. No treatment is of any avail.

Arterial Anastomoses.—Normally the retinal arteries do not anastomose. Leber[26], however, has found microscopic communications between the central artery and the arterial circle of Zinn. Schleich[27] and Seydel[28] have noted as congenital conditions cases of arterio-venous aneurisms with anastomoses between the retinal arteries.

In numerous instances, anastomoses have been observed after the obliteration of the central artery (Gonin[29], Harms[30]). In discussing retinal varicosities, Landlot's case[15] has already been mentioned. A month after the obstruction of the central artery, Coats[31] noted a communication between the superior and inferior nasal arteries, and between the nasal and temporal arteries. The question arises here as to whether new vessels really form or whether vessels already present develop and become ophthalmoscopically visible.

Venous Anastomoses.—Anastomoses between the veins are seen more often in the fundus than between the arteries. These abnormal anastomoses nearly always follow a partial or total

obstruction of the central vein (Fig. 23). Careful examination has demonstrated that these apparently new vessels arise from dilatation of capillary connections. After obstruction of the

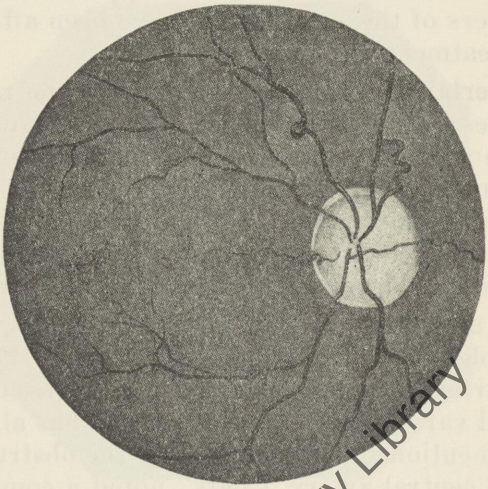

Fig. 22. Venous anastomosis following a partial thrombosis of the central vein.

central vein, a retinal vein has been observed to communicate with the chorioidal plexus by Axenfeld[32]. In another instance an anastomosis with a cilio-retinal vein has been noted (Drunn[33]).

New Formed Vessels.—After obstruction of the central vein a network of tiny sinuous vessels make their appearance. These are, however, not truly new-formed vessels, but repre-

sent, instead, the establishment of a collateral circulation by dilatation of pre-existing capillaries.

Telangiectases.—Among cases of telangiectases scattered in the literature, that of De-Wecker's[3,4] is particularly worthy of note: On the papilla and in the region about it was a dense network of veins and arteries. The maze was so close as to render it most difficult to follow any one vessel even a disc diameter from its emergence. In the affected fundus, the vessels were double the size of those in the companion eye (which was normal). In a zone of two disc diameters from the papilla there was hardly a spot on the retina not covered by vessels. This vascular abnormality was unaccompanied by any functional defect. Visual acuity was normal.

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CHAPTER IX

INFORMATION GAINED BY EXAMINATION OF THE RETINAL VESSELS

In looking at the fundus we really see not the arteries and veins themselves but the contents of these vessels. The vessel walls are normally transparent and invisible. Under pathological conditions the walls thicken and become more and more opaque. Before the thickening becomes visible, especially in cases of endarteritis, it is often evidenced by a regular or irregular narrowing of the blood column.

Changes in the Vascular Reflexes.—The light reflexes from the vessels in ophthalmoscopic examinations are derived from the vessel wall rather than from the blood column. In obstruction of the arterial circulation, as Elching pointed out, the light reflexes are still present. The veins do not present as vivid reflexes as the arteries, probably because their walls are flatter and less tense.

The light reflex disappears in advance degrees of vascular sclerosis; and it will be masked at any point along the vessel course by edema, even though very slight.

Changes in the Vessel Course.—Various congenital abnormalities in the course of the retinal vessels have already been discussed. Acquired changes are now to be considered.

Only the finest arterioles are able to change from a straight direction, and become markedly sinuous. The larger arterioles, in spite of any disorder, generally maintain their original direction. In the very fine arterial twigs, however, particularly in those about the macula, obstruction to their current causes the central portion of the vessel to become definitely tortuous. An area of hemorrhage will often show sinuous arterioles leading to it.

The veins, however, are quite subject to tortuous changes in their course. Disturbance in the venous circulation is often thus indicated. In detachment of the retina, the veins are affected not only in the involved area, but quite beyond. In the majority of cases even the examination of the disc will disclose a principal vein whose course has become sinuous. This will be found to be the vein supplying the detached area.

In obstruction of venous branches, the subsequent changes that affect the course of the vessel naturally take place peripheral to the point of interference.

Changes in Color.—Though the arterial blood is practically always the same hue, the color of the venous blood varies according to the amount

of oxygen lost and the amount of carbon dioxide carried. When the current is rapid as in vasodilatation, the venous blood is bright; when the current stagnates in the tissues the departing blood is dark. In thrombosis of the central vein, where circulatory stasis is at its maxi-

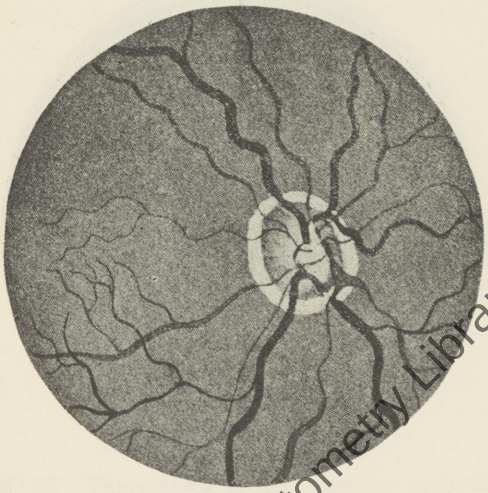

Fig. 23. Retrocession of the retinal arteries in a case of atheriosclerosis. (Hirschberg.)

mum, the venous blood is almost black. The opposite condition occurs in optic atrophy in which the difference in color between arterial and venous blood is distinctly less marked than normal.

In erythremia or Vaquez's disease, the veins

and arteries are in extreme contrast, the veins being unusually dark. But in leukemia and congenital cyanosis, the color of the vessels approach each other. In the former, the venous blood is brighter than ordinarily, though the

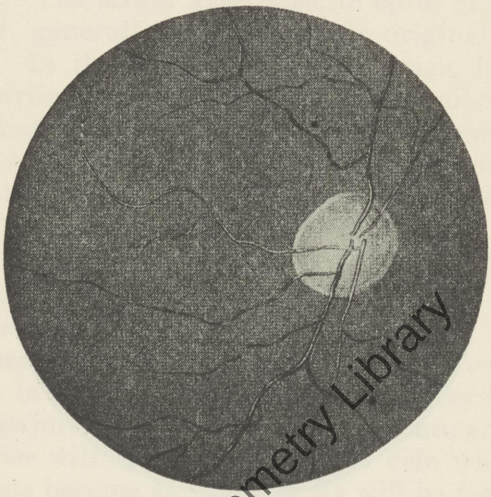

Fig. 24. Arteritis of a nasal branch.

arterial blood has its usual hue; in the latter the arterial blood has become quite dark, while the color of the venous blood has not been affected.

In detachment of the retina, the veins in the affected area and beyond are not only abnormally sinuous, but abnormally dark as well.

Finally we must keep in mind the abnormal tint of the vessels in that rare affection "lipemia retinalis" (Wagener[5] states that but 20 cases have been reported. The disc in this affection is waxy, the arteries have a salmon

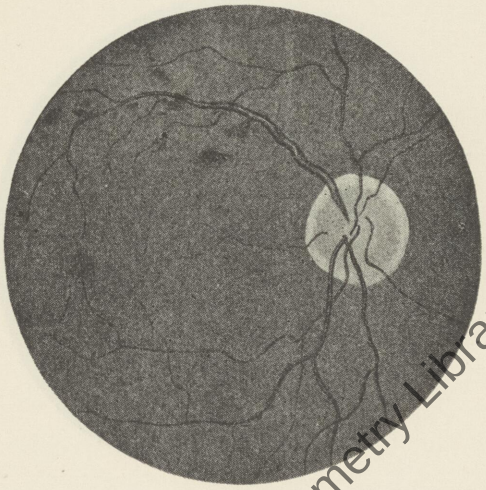

Fig. 25. Thrombosis of the superior temporal vein.

shade, and the veins are pale brown. In some cases the veins and arteries cannot be distinguished from each other[7].

Changes in Caliber.—In most cases of optic atrophy, both arteries and veins appear narrowed—especially so in the type that follows quinine intoxication. Spasm does not contract

the vessels nearly so much; in the larger vessels the effect of spasm is hardly apparent. Feigenbaum[8] noted in a case of paroxysmal visual obscuration, a dilatation of the retinal veins at the time to twice their normal caliber.

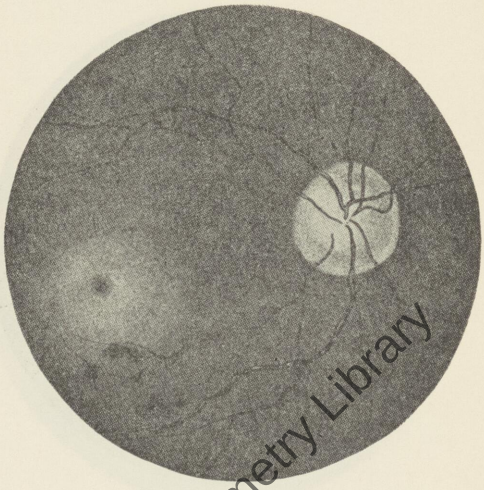

Fig. 26. Thrombosis of the inferior temporal vein.

The arteries may present at various points irregular contractions giving a beaded aspect. The latter condition is significant of arteritis, and is usually accompanied by more or less visual disturbance in the sector affected. Arteritis may determine subsequent obliteration of the vessel, or aneurismal dilation.

Arteriosclerosis may sometimes have a particularly marked effect on but one retinal artery or even on an arterial branch.

Changes in the caliber of veins occurs more frequently than similar changes in arteries.

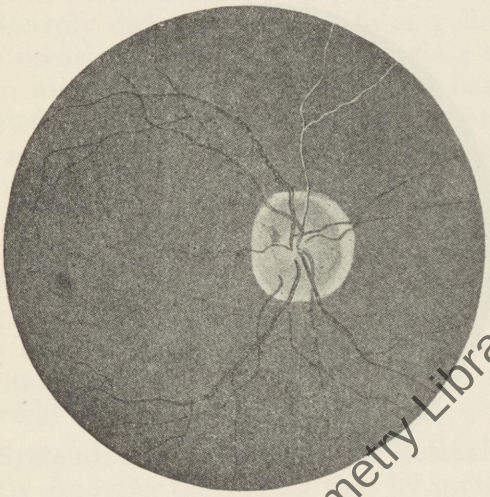

Fig. 27. Retinal arteritis. Peri-arteritic appearance.

These will be discussed later in connection with phlebitis of these vessels.

Changes in the Vessel Walls.—Under pathological conditions, the walls of the retinal vessels lose their transparency, and the vessels appear as if partly or entirely enveloped in a

white sheath. This may be exhibited on but one point of the vessel or may extend along its entire course. It is generally only beyond the limits of the disc that this condition is to be seen.

Very frequently one sees but a thin white

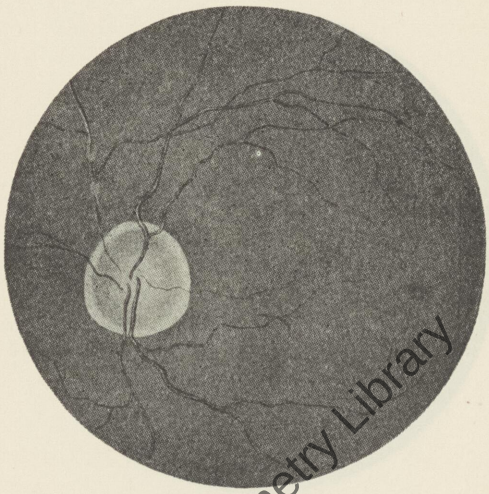

Fig. 28. Arteritis of the superior nasal branch.

line along each side of the affected artery. As the vessel approaches the periphery, these lines become relatively more prominent and cause the blood column seemingly to disappear.

These changes characterize especially the lesions of pariarteritis which may exist for quite a time without completely arresting the

circulation—even when the vessel appears replaced by a fibrous cord.

The walls of the retinal veins are but rarely subject to similar modifications. Harms[1] has reported a very interesting case of juvenile retinal periphlebitis, in which the vein for a short stretch seems replaced by a whitish strand.

Sharply defined, small white or yellow spots are occasionally seen on the arteries; very seldom along the veins. These have been interpreted as calcareous concretions (Adams), and as areas of fatty degeneration (Manz). The process aligns itself with that rather rare affection of the systematic arteries called "nodose arteritis" (Roger and Gouget[2]). Histological examination of these nodules shows a thickening of the inner coat; a hyaline degeneration in the middle coat; and an infiltration in the external tunic, which may attain to ten times its normal thickness.

Systemic Examination of the Retinal Vessels.—By studying the fundus first by indirect ophthalmoscopy, one follows the vessels best from the disc to the periphery. Details are then closely observed by the direct method. It is essential that the peripheral as well as the central portion of the field be inspected.

Ophthalmoscopy with the red-free light is of great help, especially for determining with certainty areas of edema and hemorrhage.

Finally by noting the effects of ocular compression, we learn the pressure relations of the retinal vessels, and gain an idea of the tenseness of their walls.

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CHAPTER X

AFFECTIONS OF THE RETINAL ARTERIES

Though the retinal arteries are subject to the same pathology as vessels elsewhere, they deserve special attention for nowhere else in the living being can the morbid processes be so well studied.

The terms "arteritis" is inclusive of all arterial lesions whether inflammatory or toxic; whether based on systemic dyscrasiae, or secondary to hypertension. Some have given to arterial lesions a predominant importance, attributing to poor blood supply, the invasion of organs by connective tissue; others believe that parenchymatous changes are not simply secondary to lesions of the vessels, but that both are produced simultaneously by the same cause. Letulle and Nattan-Larrier[1] write: "As long as the caliber of the artery remains unaffected, the parenchyma experiences neither nutritive nor degenerative changes, and whatever symptoms occur, even though severe, are of functional origin.

Arteritis may be more or less localized. Castaigne and Esmein[2] cite chronic alcoholism and chronic lead poisoning as examples in the course of which but one arterial region may be affected. Functional disorders first develop in

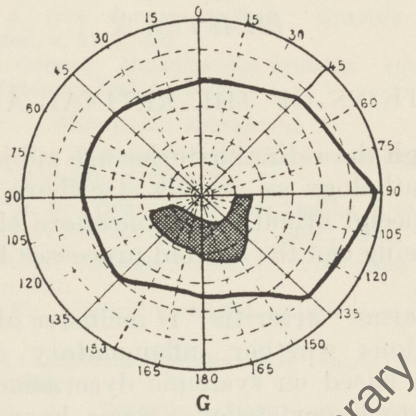

Fig. 29. Paracentral scotoma in a case of retinal arteritis.

the part affected; then chronic lesions of the arteries particularly those of the sclerotic type.

Physical Signs of Arteritis of the Retinal Vessels.—The various physical signs that characterize lesions of the retinal arteries have been mentioned in the preceding chapter. In endarteritis and periarteritis the previously invisible vessel walls become visible. Sclerosis may affect either the entire course of a vessel, or only a sector thereof; it may be evident in

islands; or in but one or several spots. As the arteritis progresses, the caliber of the vessel narrows; or the wall may yield at some point and form an aneurismal pouch. Proliferative

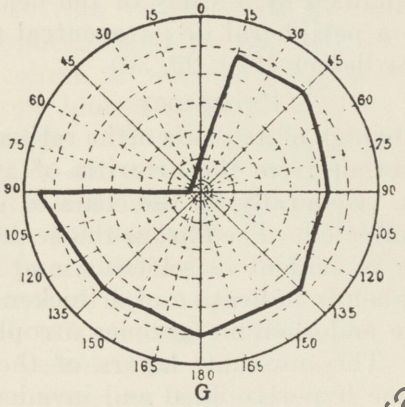

Fig. 30. Obliterating arteritis of the inferior nasal artery.

endarteritis often produces irregular contractions, and a consequent beaded aspect.

Added information is obtained from a study of the induced arterial pulse. The more sclerotic a vessel, the feebler and more limited its reaction.

Symptoms.—The subjective sensations that accompany arteritis of the retinal vessels vary, of course, with the nature and localization of the lesion. An ateritis of the central artery produces naturally a more serious functional

disturbance than an obliteration of a peripheral arteriole. When a lesion is localized to a branch vessel, the central visual acuity may remain unaffected. Visual discomfort exists, however, and is explained by a study of the fields which will show a peripheral or paracentral scotoma, or a sector defect (Fig. 29, 30).

Pathology

The pathology of arteritis ofthe retinal vessels is no different from the arteritis of arterioles elsewhere. Generally all the coats of the arteriole are affected. The fibrous thickening of the adventitia is evident in sclerosis, and the contracted lumen is indicative of a thickened inner coat. The endothelium becomes atrophied and lamellar. The muscular layers of the middle coat become hypertrophied and invaded by fibrous and hyalin tissue. The proliferation of these coats may end in effacement of the lumen of the smaller arterioles, which thus become transformed into rigid cylinders.

Calcareous plaques, so often noted in larger arteries, are hardly ever seen on the arterioles. In luetic arteritis the endothelial proliferation is more active, leading more rapidly to vascular obstruction, and the complications of thromboarteritis.

Microscopic lesions of the retinal vessels have been observed by Hertel[3] in subjects that in life never presented any visual complaint. This

would be expected, for as long as the arteries and capillaries are permeable, the visual function is conserved. Hertel chose for his study 14 victims of systemic arteriosclerosis, who were free from visual disturbances, but who showed ophthalmoscopic findings such as serpentine course of vessels, irregularity of caliber, narrow lumen, white sheaths, or incipient optic atrophy. In one group, diffuse lesions covered the entire extent of the vessels; the external tunic showed an overgrowth of connective tissue, but the inner coat was normal. In the other group the lesions were localized in certain areas, and the inner coat was considerably thickened (six to eight times that seen normally). Vessels that were usually 170-180 mu in caliber were as a consequence narrow to about 90 mu.

Etiology

According to Castaigne and Esmein, acute lesions of arteritis are generally found superimposed upon an arteritis of the chronic type. The experimental pathologist seldom can produce an acute arteritis by the simple introduction of virulent bacteria into the circulation, but success follows if the artery has been previously injured.

However, young individuals may become affected with acute arteritis in the course of acute infectious diseases. The retinal arteries have

been thus affected in mumps, acute articular rheumatism, malaria[4], influenza, and scarlet fever.

But arteritis is usually a chronic affection of slow evolution—based on a chronic intoxication or a chronic infection. Among determining factors are lead poisoning, alcohol and tobacco addiction, intestinal autointoxication, uremia, and diabetes. Most potent of all is syphilis. Ateritis may be manifest in any stage of this disease including the congenital form. Not only is syphilis dominant in the production of localized arteritis in the aorta, coronary artery, cerebral arteries, and elsewhere, but often it is the basis of the diffuse lesions of arteriosclerosis that seem to be but the accompaniment of senility. Age, however, is itself predisposing, most of those affected having passed the fifth decade.

Hypertension also may favor arteritis. However, the cause of the hypertension may often be lues. In two cases of arteritis with retinal hemorrhages in my experience specific treatment gave a remarkable result in spite of a negative blood Wassermann.

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CHAPTER XI

COMPLICATIONS OF RETINAL ARTERITIS

The principal complications of retinal arteritis are hemorrhage and obliteration.

Hemorrhage From the Retinal Arteries

Hemorrhage in the retina generally occurs from capillaries and veins; rarely is an artery the source. No one has directly observed a rupture of the central artery. True, such an observation would be ophthalmoscopically impossible, as the hemorrhage would flood the vitreous, or detach the retina. Still, since arterial hemorrhage occurs in the brain, it is quite possible that certain retinal hemorrhages are also of arterial origin. Such a hemorrhage would stop rather rapidly, as the blood-pressure in the smaller arteries is only a little greater than the chamber pressure. Since the first effect of hemorrhage is to increase the intra-ocular tension, an equilibrium is soon established and further hemorrhage ceases. When the eyeball is opened by trauma or operation, no such check comes into play, and an expulsive hemorrhage may then follow.

Obliteration of the Retinal Arteries

A retinal artery may become obliterated by either arterial lesion, embolus, or spasm. Gradual obliteration may follow the increased thick-

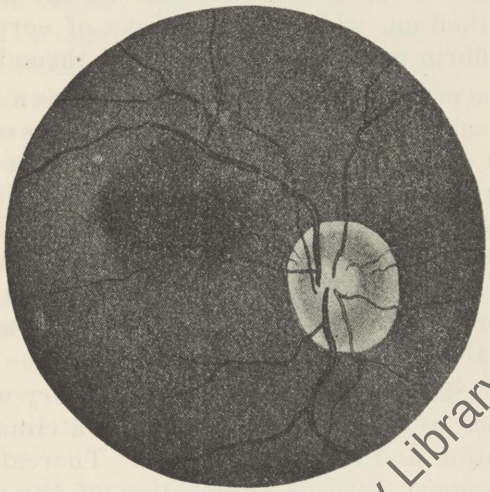

Fig. 31. Sub-muscular hemorrhage, probably of arterial origin. A grayish, edematous zone will be observed on the path of the superior temporal artery, of which the hemorrhage seems to be a part. Note, also, the condition of the two small macular arteries. (Woman, aged 57. Sudden onset. Arterial hypertension. Followed by complete cure.)

ening of the walls incident to arteritis. The circulation becomes arrested in the peripheral arterioles near the capillary bed. The larger arteries are not affected by this process except secondarily.

More often obliteration is induced by thrombus formation sequential to an "obliterating endarteritis." The arterial lesion and the constricted lumen greatly favor the production of thrombus. A coagulum forms on the affected endothelium, which by additions of corpuscles and fibrin ends in an obliterating thrombus.

The endarterial lesion that precedes a thrombus may be determined by the infectious or toxic state of the circulating blood. Bacteria attach themselves to the endothelium; and they, as well as the disorganized protoplasm, are in turn attacked by the leucocytes in the periphery of the blood stream. The so-called marasmic thrombi that form in cachectic conditions have probably a similar origin.

In 1859 embolus of the central artery was described by Von Graefe[1], who gave a clinical description and autopsy findings. Thereafter almost every case of obliteration of the central artery was thus interpreted.

Haab, and later Harms, showed that thrombosis occurred more often than embolus. Rohmer[2] stated that it would be indeed surprising if emboli left the heart and great vessels so often to lodge in an organ so distant and so isolated as the eye. The ophthalmic artery leaves the internal carotid at almost a right angle; and the central artery of the retina is at a similar angle to the ophthalmic. Such conditions must

be considered quite unfavorable to the circulation of emboli.

Whence the origin of the emboli that do occur? Though emboli for the most part develop in the venous system, these but very rarely can pass through the pulmonary circulation to reach the retina. Walter[3], however, encountered such a complication in a case of "phlegmasia alba dolens"; and Stuelp[4] in a patient with uterine phlebitis.

The chief source of retinal emboli is endocarditis, acute or chronic, and particularly the valvular lesion of mitral stenosis. Plaques of athermatous arteriitis that become free in the circulation are another frequent source of arterial emboli. When such debris plugs a retinal artery, it may possibly have a nearby origin, such as the internal carotid or the ophthalmic artery.

Fat emboli or air emboli but rarely affect the central artery[5]. Certain ocular accidents following the subcutaneous injection of paraffin in the nasal region have been reported as emboli of the central artery, but according to Rohmer[6] it is likely that these have really been cases of venous thrombosis.

Addario la Rocca[27] noted a case of embolus of the central artery after an injection of arsenobenzol. But it may be that the disease rather than the remedy is to be incriminated.

The central artery may receive a septic embolus, and panophthalmitis result therefrom. Candian[28] reports such a case. A man, aged 40, had an infected lower molar extracted. Three days later the vision in one eye became totally lost. After that there appeared ciliary injection, iritis, hypopyon, and clouding of the vitreous.

Symptoms of Retinal Arterial Obliteration

When an embolus is arrested in a limb, violent and prolonged pain follows; but due to the specialized sensibility of the retina no pain is experienced in the obliteration of the central artery. Sometimes, however, the advent of blindness is accompanied by an uncomfortable sensation. A patient under my observation went to sleep with excellent vision; he woke up suddenly with the feeling that he had become blind. On testing the vision with a lighted candle he discovered that he had totally lost the sight of the right eye. The ophthalmoscopic picture the next morning was that of an acute obliteration of the central artery. Is it not probable that the patient had been aroused by some painful sensation and his attention thus called to the condition of his eye?

Still if the unaffected eye be healthy, the patient does not realize generally for some time what has happened. He has the sensation of a veil before his eyes; he rubs them; and it is

only after closing the healthy eye that he discovers a more or less complete loss of vision in its companion. If the obliteration affects the trunk of the central artery, the blindness will be total; but if a branch only is involved, a sector defect results.

The arterial obliteration and consequent blindness may supervene at any time, asleep or awake, at rest or during exertion. Careful questioning may reveal that for some while previously the patient has had certain prodromal sensations. A sort of fog perhaps would momentarily come before the eyes from time to time. Such a history would be indicative of previous disease of the artery, and would speak for the obliteration being due to thrombus rather than to embolus. Similarly, in "intermittent claudication" of the leg, disorders of sensation and function announce the advent of thrombosis and gangrene.

Such prodromes are probably due to arterial spasms incited by the endarterial lesion present. Wagenmann[7] and Hoppe[8] have reported interesting ophthalmic cases bearing upon this point. In true embolus, the blindness comes on suddenly and there are no prodromal sensations.

Ophthalmoscopic Signs.—In acute obliteration of the central artery, the vessels are reduced in caliber—the arteries much more so

than the veins. The disc is blurred, and a milky whiteness extends from it, especially towards the macular region; but the macula itself stands out in contrast as a distinctly outlined cherry-red spot.

(a) The Vessels.—If the obliteration affects the central artery no arterial pulse can be induced by ocular compression. Occasionally in the peripheral arterioles, segmentation of the blood column has been noted.

In the veins this segmentation of the blood column occurs shortly after obliteration, and extends to the large papillary vessels. After the vis a tergo from the arterial pressure is suppressed, the blood column would remain immobile were it not for thoracic aspiration. The suction force, however, is not strong enough to empty the vessels, but draws the blood column away little by little, and thus is caused the segmentation. The force of the intra-ocular pressure likewise helps generally to empty the veins; but if the thoracic suction is quite weak, and the veins difficultly permeable a retrograde movement may be produced.

(b) The Retina.—The retina is early affected. An examination made just after the advent of blindness will already reveal some retinal cloudiness. This cloudiness, however, passes away by the end of the first week. Pathological examination has demonstrated that the milky ap-

pearance of the retina is due, not to edema, but to incipient degeneration of the nerve fibers and the ganglion cells. This reaction is seen to some extent even in the partial obliteration that occurs in angiospasm, and produces the whitish trail seen along each side of the contracted vessel.

In complete obliteration of the central artery the retinal cells are functionally dead in a few hours. After the destruction of these most delicate elements, the atrophy proceeds very slowly, and the ophthalmoscopic signs become similar to that in optic atrophy of other causes.

(c) The Cherry-Red Macular Spot.—The cherry-red macular spot occurs almost constantly in acute obliteration of the central artery. The size thereof varies from one-seventh to one-third of a disc diameter. It is not seen as early as the retinal changes. In a case under my observation from the first hour of the advent of blindness (due to embolus), the spot did not appear until the tenth hour. In a case of Forster's the spot was seen at the end of the eighth hour. The spot passes with the disappearance of retinal cloudiness, sometimes earlier.

The appearance of the spot is due simply to a matter of contrast. The choroid, which maintains its normal color, is seen through the macular window, and stands out clearly against the

whitish background of the retina. The color may vary from a bright red to a dark reddish-brown; the variation may be due to the relative degree of edema in the retinal cells.

The Reestablishment of the Retinal Circulation.—In the great majority of cases a reestablishment of the retinal circulation has occurred in a few weeks after the obliteration has taken place. The veins no longer show a segmented blood column, but are normally filled; and ocular compression provokes once more an arterial pulsation.

I have found the filling of the veins to be the first change to occur. In one of my cases the venous circulation was reestablished by the fifth day; in another case the vessels had recovered their normal appearance by the second day. In a third case, seen in Vaquez's service, in which the obliteration was due to an embolus of mitral origin, both the venous and arterial circulations were completely reestablished in seven days. Harlan[8] reported a case in which in two days the arteries though small were again permeable.

One can in some instances hasten the restoration of the retinal circulation by the aid of amyl nitrite. The following case is illustrative:

Thrombosis of the Central Artery, Right Eye.—The patient, 48 years old, was in apparently good health. For the previous two years he had occasionally experienced a momentary hazi-

ness before the right eye. For the past several days there had been a sensation of discomfort in the use of the left hand.

On Dec. 28, 1920, at 8:00 a. m. he suddenly felt that the right eye had become completely

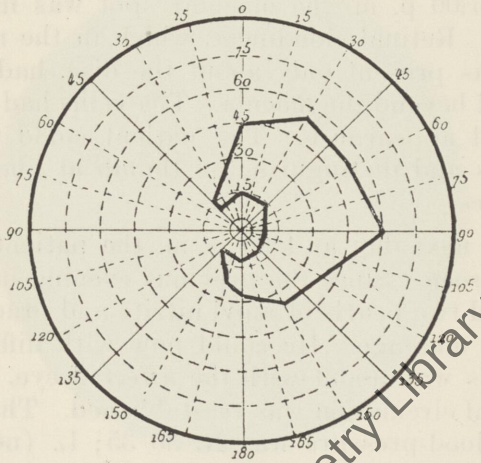

Fig. 32. Visual field several days after the onset of obliteration of the trunk of the central artery. A temporal section alone is preserved.

blind. At 10:00 a. m. he came to the clinic for examination. Vision was nil. The fundus had the typical aspect of acute obliteration. The venous column was segmented. No arterial pulse could be provoked. At 10:30 a. m. the patient was given amyl nitrite to inhale. Marked

venous dilatation immediately followed. The blood column in the veins became reestablished. Hand movements could be seen, and light had a violet appearance. No macular spot had yet appeared.

At 5:00 p. m. the macular spot was in evidence. Retinal cloudiness, which in the morning was present only about the disc, had now spread beyond the macula. The veins had their normal appearance. The patient could count fingers and distinguish the thumb at one foot distance.

The next day at 1:00 p. m. the patient was again seen. Since the previous evening he had inhaled two pearls of amyl nitrite and practiced ocular massage. He could now with difficulty find his way about with the affected eye. The arterial circulation was reestablished. The retinal blood-pressure was R. 30/35; L. (normal eye) 35/70.

On Dec. 30 at 10:00 a. m. he learned of the sudden death of his sister from cerebral embolus. He was profoundly affected. All at once he felt some pain in the right orbital region, and the blindness became again as complete as on the first day. When seen at 5:00 p. m. retinal cloudiness was present, narrowed pulseless arteries, and fragmented venous column. The vision was again nil. A systematic examina-

tion disclosed no cardiac lesion. Amyl nitrite was continued three times daily.

On Dec. 31, a slight amelioration was noted; tramway rails could be seen.

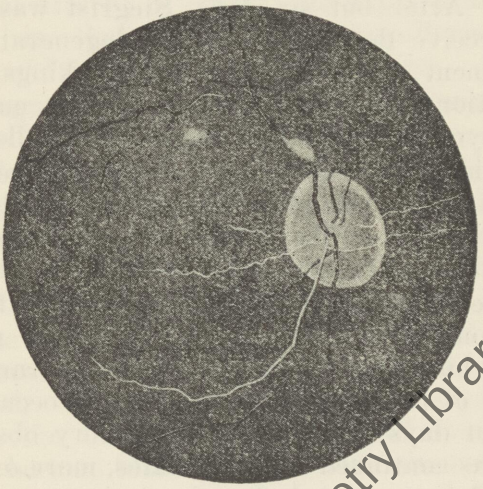

Fig. 33. Obliterating arteritis supervening on a thrombosis of the central vein.

On Jan. 5 the retinal cloudiness and the macular spot were still marked.

By Jan. 11 the vision was not further improved, except in a small region temporarily, where hand movements were perceived. The arterial current was again established. The blood pressure of the central arteries were: R. 35/70, L. 40/80.

The Consequences of Arterial Obliteration.—The loss of blood supply induces atrophy of the nerve-fibers and of the ganglion cells. Elschnig and Von Nuel noted that the atrophy was considerably advanced by the fifth or sixth week. After but six days, Siegrist was able to perceive definite signs of degeneration—effacement of nuclei and cell markings, and formation of vacuoles, homogeneous masses, and myolene droplets in the nerve bundles.

Clinical observations have demonstrated that after six or eight hours of obstruction the re-establishment of the circulation will bring but a partial return of visual function.

Experimenting on the rabbit, Baquis[11] found that compression of the central artery maintained but for 15 minutes produced transient lesions of the ganglion cells (healing occurring in eight days); but if the circulatory obstruction was continued for 30 minutes, more or less marked permanent destructive lesions resulted.

R. Sand[12] has shown that total arrest of the cerebral circulation cannot be tolerated by mammals for more than 3 to 25 minutes. After this period the brain can still be revived, but with resultant serious functional disorders and pathological changes.

The prognosis of acute obliteration of the central artery is indeed grave. The vision remains lost, except in the rare cases (probably

due to arterial spasm) where the circulation was very early re-established. Occasionally a luminous sensation persists in a portion of the temporal field (Fig. 30). In some cases this is apparently due to the existence of a cilio-retinal artery; in others, the explanation perhaps is that the nutrient capillaries are derived from beyond the obstruction.

Frequency of Acute Arterial Obliteration.—Acute obliteration of the central artery occurs rather infrequently. In Hirschberg's clinic there was one to every 1,300 eye cases. Dufour and Gonin in the hospital at Lausanne noted one to every 2,400 patients; Schöbl, one to 5,000; Wuttich, one to 10,000.

The two eyes may become affected simultaneously, or after a more or less intervening period. Harms has collected 20 such cases from the literature. In three of these both eyes became blind within a few minutes. Van Duyse's case concerned a man aged 71 with mitral insufficiency, who on leaning forward was in three minutes affected by bilateral blindness.

Fisher,[14] in a study of 200 cases, finds that the right and left eyes are statistically about equally affected. He points out, however, that cerebral apoplexy affects mostly the left side, because the left cartoid for anatomical reasons is more exposed to emboli. It seems hence

probable that if the statistics included only those cases of acute obliteration due to emboli, they would show a predominant incidence on the left side.

Obliteration of Branches of the Central Artery.—Should the obliteration affect a branch artery, the ophthalmoscopic examination will definitely show arterial contraction, and a milky retina in the involved zone. The visual field will show a blind sector corresponding to the artery affected. Any branch may suffer; in several observations[15,16] a cilioretinal artery has been obstructed, with a resulting central scotoma.

The Nature of the Obliteration.—The principal ophthalmoscopic signs of thrombus and embolus are the same. Patients affected with thrombosis usually have hypertension and often give a history suggestive of previous local or systemic arterial disturbance.

Embolus is most generally derived from a mitral lesion. A sudden attack of blindness coming on one thus affected would hence most probably be due to embolus. The embolus can be but rarely actually observed. Hess[17] examined a woman with heart disease, 54 years old, 10 minutes after the loss of vision. After massage, the embolus became visible at the origin of the superior branch as a tiny white cylinder. Further massage caused the embolus to

advance to the next bifurcation. The following day the embolus was absorbed and the vision was 20/25.

Sometimes there is seen on the disc, just below the embolus, a sacciform aneurysmal dilatation of the trunk of the central artery. This aneurysm, which pulsates on ocular compression, disappears in several days.

Chronic Obliteration of the Central Artery of the Retina.—As a result of endarteritis the blood supply of the retina becomes progressively more and more reduced. The retina becomes pale, the vessels narrowed, and the pulse reaction to ocular compression feeble. The difference between the retinal systolic and diastolic pressures is definitely less than normal. The veins are also narrowed, and the color of the venous blood approaches that of the arterial; the venous pulse can no longer be induced.

In more advanced cases white sheaths encircle the arteries. The inhalation of amyl nitrite in these cases will for an instant improve the precarious circulation.

The disc is more or less pale; this atrophy is due to similar lesions of the vessels supplying the optic nerve. The disc may also become excavated. Schnabel claims that excavation of the optic disc is essentially due to the formation of small gaps in the interior of the nerve. Moray[1] has elaborated this conception. He

believes these lacunæ are formed as a consequence of the anemia resulting from vascular obliteration. In glaucoma this anemia is induced by the compression of the capillaries of the optic nerve and retina.

Functionally, the vision becomes less and less; the visual field becomes contracted, and small central and paracentral scotomata appear. Rarely does absolute blindness supervene. The inhalation of amyl nitrite, in improving the circulation, produces a transient subjective improvement in vision.

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CHAPTER XII

AFFECTIONS OF THE RETINAL VEINS

The early stages of phlebitis are seldom observed ophthalmoscopically. The inflammation of the venous walls is rarely recognized before complications such as rupture or obliteration have occurred. The venous circulation may be a long time defective before revealing any ophthalmoscopic changes, or occasioning any subjective disturbance. Even the obliteration of the large venous trunks does not produce symptoms at all comparable to that of obliteration of corresponding arteries. In acute phlebitis, moreover, it takes several days for venous lesions and their complications to appear.

In the early stages of acute phlebitis a slight edema about the affected vessel may sometimes be noted. The vein central to the lesion tends to appear hazy and abnormally light colored, while beyond the lesion it becomes darker than the veins about it.

The chronic forms of phlebitis in the retina, as in phlebitis elsewhere in the limbs and vis-

cera, are most often of syphilitic or tuberculous etiology.

In a rare case of Harms, sclerosis affected almost all the retinal veins, particularly at the

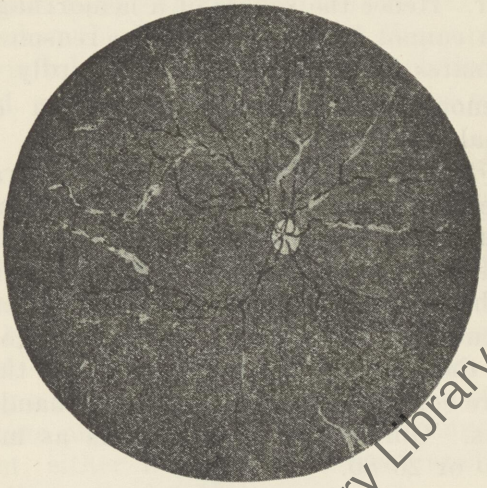

Fig. 34. Juvenile periphlebitis. (Harms.)

periphery. This condition was noted in both eyes in a young man of 19, and was accompanied by retinal hemorrhages.

Rupture of Retinal Veins.—Most hemorrhages come from the venous end of the capillary bed. It is rare to observe, either with the ophthalmoscope or microscope, a rupture of the vein itself.

Venous hemorrhage, like arterial, is quickly

arrested by the increased ocular tension it produces. Since the pressure in the small veins is greater than in the large ones, hemorrhage occurs more readily from them than from the latter. Hence the source of a hemorrhage very often cannot be located for the reason that it originates from a vessel that is hardly visible.

Venous Obliteration.—Obliteration of the retinal veins occurs rather frequently. In the days preceding thrombosis of the central vein there are generally no prodromal symptoms. Abruptly, in the course of a few hours or over night, the vision becomes very hazy. There is not, however, a sudden total loss of vision. The patient may not be able to read or go about with the affected eye, but even under the most severe conditions he is able to note hand movements. Quite often the vision is as much as 20/60 or 20/80.

If the patient complains of headache or periorbital heaviness, it may mean that the phlebitis has extended posteriorly beyond the scleral plate. But on the other hand inflammatory conditions in the environs of the orbit may have been the determining cause of the thrombosis of the retinal vein, and be the occasion for these symptoms.

The reason that the visual function is to some extent conserved is because the thrombus generally only partially obliterates the caliber of

the vein, and a certain degree of circulation still goes on. Even when the obstruction is complete and the blood current strikes a venous cul-de-sac, it must be remembered that the capillaries have still received the arterial blood. Only when the lesions spread to the capillary and arterial bed is the capillary circulation suspended, and with it the nutrition of the retinal cells.

The Ophthalmoscopic Signs of Obliteration of the Central Vein.—The ophthalmoscopic picture varies according to the time after the obliteration that the fundus is examined. In the first day the disc is congested and its outlines blurred. The veins near the disc are dilated, tortuous, and very dark. Often after making a curve, a vein disappears for a stretch in a zone of edema. Retinal hemorrhages are present, either isolated or disseminated over the entire retinal field.

As the days pass the condition becomes more marked, the disc becomes redder, the hemorrhages more numerous. About the disc the retina is almost covered with hemorrhagic islands. The hemorrhages are visible in the macular region and up to the extreme limits of the retina, though in the periphery they are less confluent. Sometimes the hemorrhages thin out, and seem to follow the axis of a vessel; then again they occur in lakes and

puddles. The more recent the hemorrhage, the brighter red the color. A whitish or grayish cloud, indicative of retinal edema, is often present, especially in the macular region.

The arteries on the disc are buried in edema, and are consequently very indistinct, sometimes entirely invisible. They would seem obliterated, were it not that ocular compression induces their pulsation. The veins are hazy, and sometimes markedly dilated, the dilatation being best seen, however, beyond the disc. Parts of the veins are so engorged and so black as to suggest the appearance of blood-filled leaches. The sinuous veins become lost every so often in a zone of edema, to reappear again a little further on.

The venous circulation rarely becomes re-established; generally the obliteration is permanent, and extends gradually to the entire retinal circulation. The main veins, at first so dilated, become narrowed more and more, and finally appear but as white cords. The arteries are visible again with the disappearance of the papillary edema, but are contracted and immobile. The optic nerve becomes atrophic. In this terminal stage, one may see very frequently on or about the disc, tiny sinuous vessels—anastomoses between the chorioidal and retinal circulation—which, though normally invisible, become dilated to a maximal

degree by the obstruction of the venous circulation.

Thrombosis of a Branch Retinal Vessel.—The symptoms of thrombosis of a branch vein

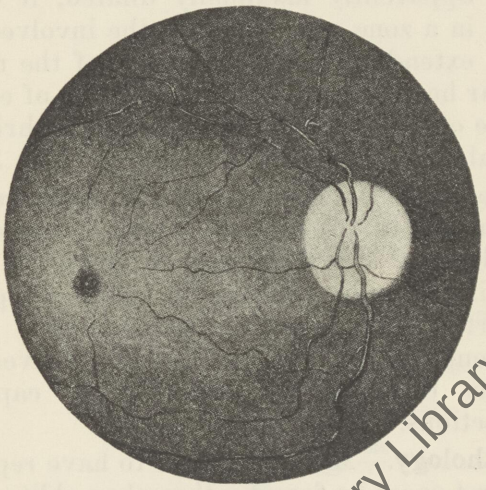

Fig. 35. Thrombosis of the inferior branch of the central vein. Onset. Edema of the macular region. Observe the appearance of the vein, and the sharp difference of coloration which undoubtedly marks the thrombotic point. There is no longer any hemorrage.

vary naturally with the branch involved. If only a peripheral vein is affected, the visual disturbance is relatively slight and localized to a sector of the visual field (Fig. 35). But if a macular vein is thrombosed, central visual acuity will be markedly lessened, and an abso-

lute or relative central scotoma will exist.

The vein central to the obstruction is dilated, dark, and sinuous. This appearance becomes more marked as the disc is approached, until, when apparently maximally dilated, it disappears in a zone of edema. In the involved segment, extending to the periphery of the retina, appear hemorrhages, and often areas of edema. In one of my cases (not affected by nephritis) a typical macular star was observed (Fig. 37).

The return to normal—though quite rare in thrombosis of the central trunk—is not exceptional when a branch only is involved. The vein, if it remains obliterated, becomes replaced in part of its course by a whitish cord. Often anastomoses seem to establish themselves, due to the dilatation of pre-existing capillary connections.

Pathology.—Michel appears to have reported the first case; he found a thrombus obliterating the central vein posterior to the scleral plate for a length of ½ mm. The level of the scleral plate, or a little posterior, is the site of election for the formation of these thrombi. This localization is favored by the constriction of the vein here, and the traction of the ocular movements. Coats remarks that an obstruction occurring more posterior would give no symptoms because of the rich venous anastomoses in the optic nerve. The obliterating mass is at

first structureless, and composed of fibrin and some blood corpuscles. Little by little, the plug organizes and retracts, losing for some extent at least, contact with the venous wall.

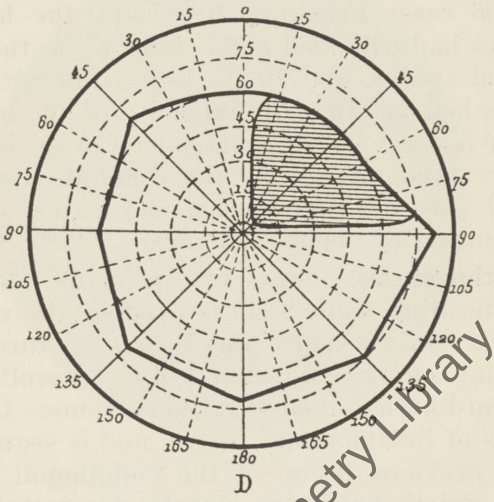

Fig. 36. Phlebitis of the inferior nasal branch of the central vein. Recovery. Vision, 20/25. A relative scotoma persists four months after the onset. (Woman, 63 years. Arterial hypertension. Had already had three attacks of phlebitis in the lower limbs.)

Gonin[2] remarks that the obliteration commences at the precise point where the vessel becomes indistinct in the ophthalmoscopic picture, and is prolonged centripetally for a dis-

tance difficult to determine, as the emptied vein below the obstruction is hardly visible.

The hemorrhages commence and are of maximal degree in the internal coats of the veins, from which it may extend beyond. In the 36 cases examined by Coats, the hemorrhages had extended eight times up to the subretinal spaces, and 10 times into the vitreous. Coats believes that the intensity of the hemorrhage is less when an endarteritis of the central artery accompanies, as the diminution of arterial pressure thus occasioned means also a diminution of venous pressure.

Pathogenesis.—Only exceptionally does a proliferating endophlebitis produce the venous obstruction. Usually this is due to thrombus forming on the site of a lesion. Letulle and Nattan-Larrier[3] hold that every venous thrombus is of inflammatory origin, and is sequential to a previous lesion of the endothelial layer. This endothelial lesion may be due to tuberculosis, syphilis, or acute infections, such as rheumatism, typhus, or phlegmasia alba dolens. Previous chronic lesions may be predisposing, as well as toxic agents (alcohol, lead, and phosphorus), and constitutional dyscrasias (gout, diabetes, and chronic nephritis).

The division of the central vein at a right angle on the disc predisposes anatomically to thrombus formation as it tends to promote

stagnation of the blood current. The crossing of a vein by a thickened artery may produce obstruction and subsequent thrombosis.[8]

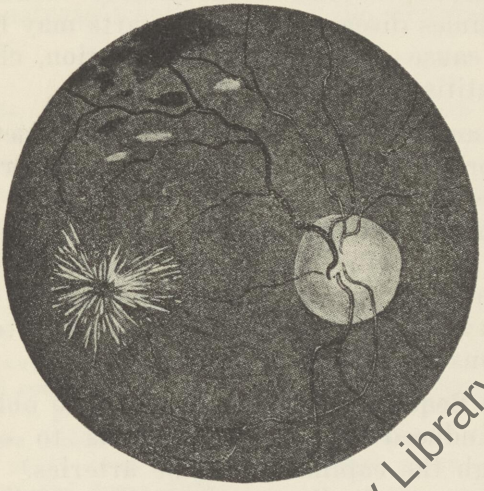

Fig. 37. Thrombosis of the superior temporal vein. Macular star. (Woman, aged 44. Nojalbumiuric, non-azotemic. Arterial hypertension. Service of Dr. Morax.)

Etiology.—Thrombosis of the central vein is somewhat more often encountered in women than in men, and most often between the 55th and 65th year. Associated disease of the veins elsewhere in the body may be present, such as hemorrhoids, varices, and phlebitis of the extremities. Thrombosis may occur as an acute lesion complicating sinusitis, orbital cellulitis,

erysipelas, meningitis, and influenza (Jackson[4]).

Chronic conditions are, however, generally responsible, as has been already mentioned. Sometimes disease in distant parts may be the basic cause—such as dental infection, chronic prostatitis, and chronic metritis.

Traumatism may be a determining factor in the production of phlebitis of the retinal vessels as well as elsewhere. Four of my cases gave a history of minor blows on the eyes a short time before the advent of their trouble. This may be, of course, but coincidence. Cases with similar histories have been reported by Everbusch,[5] Wiser,[6] and Wibaut.[7]

The Sequelae of Thrombosis.—The obliteration induced by thrombus tends to extend through the capillaries to the arteries. Optic atrophy may finally ensue. But sometimes the venous circulation becomes more or less re-established. With one-sixteenth of the venous caliber open, the retinal function may still be conserved. The later development of a collateral circulation is of no avail, however.

Hemorrhagic glaucoma frequently follows thrombosis of the central vein. Of the 36 cases examined by Coats, 35 had been enucleated for this complication.

Diagnosis.—Occasionally obliteration of the

vein is mistaken for that of the artery. The ophthalmoscopic pictures are, however, quite distinctive. In case of doubt, the response of the arterial pulse to ocular compression would demonstrate whether or not the arteries were patent.

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CHAPTER XIII

RETINAL HEMORRHAGES

Retinal hemorrhages are a symptom of numerous abnormal conditions, both local and systemic. Quantitatively retinal hemorrhages are almost negligible, but because of the extreme differentiation of the retina the retina is unable to tolerate that which would be innocuous to other tissues.

The Ophthalmoscopic Appearance of Retinal Hemorrhages.—Sometimes the difference in color gives a clue to the origin of a retinal hemorrhage. When fresh, the darker hemorrhages are generally venous, the lighter arterial. Most often, however, the color gives information as to the age of the hemorrhage; the older the case, the darker the color.

The number of hemorrhages and their distribution is quite variable. In albuminuric retinitis the typical location is about or near the disc.

The form of a hemorrhage is largely determined by its situation in the retinal layers.

When flame-shaped, the hemorrhage is in the nerve-fiber layer, the position between the nerve bundles causing this distinctive shape. Stippled hemorrhages occur most often in the macular region. This is a type frequently encountered in senile vascular lesions, and is difficultly absorbed. Most important are the puddle-like hemorrhages, such as are found in albuminuric retinitis and in thrombosis of the central vein. These occur mostly about the disc or near a large venous trunk. Sometimes an isolated puddle covers the macular area. Often these various types of hemorrhage appear simultaneously.

Special forms of hemorrhage are the retro-retinal and pre-retinal types. In the former, the hemorrhage accumulates between the rod and cone layer and the pigment epithelium. The origin is either retinal or choroidal. When profuse the appearance of retinal detachment is sometimes simulated. Generally these hemorrhages are more or less circular, are lighter than the usual hemorrhage, and, most important for diagnosis, are traversed by intact retinal vessels.

In the pre-retinal type, the hemorrhage forms between the retina and the vitreous. Merigot de Treigny distinguishes two varieties: (a) That located really outside the retina, between the hyaloid and retinal membranes; (b)

that in the retina, between the nerve-fiber layer and the internal limiting membrane. These hemorrhages generally are not far from the disc, and very often in the macular region. They appear like a red pocket, whose shape varies somewhat with the position of the head, but whose upper limit is always a horizontal line. These pre-retinal hemorrhages may appear in any hemorrhagic affection of the retina and have no special diagnostic significance. It is probable that in the majority of cases the origin is from the retina, and the extent speaks for an arterial source. Occasionally the hemorrhage breaks through the hyaloid membrane and floods the vitreous.

A venous hemorrhage is sometimes limited by the vascular sheath, and so forms a sleeve of variable length about the vessel. As absorption goes on, the hemorrhagic mass breaks up into separate particles, making the vessel appear then as if strung with beads.

The Sequelæ of Retinal Hemorrhages.—Hemorrhages in the retina do not go through the color changes that usually characterize ecchymoses elsewhere. Their color remains always reddish. They may be either entirely absorbed or leave traces behind. Isolated hemorrhages, and especially pre-retinal hemorrhages, are the most completely absorbed; the bright red becomes brownish, healthy retinal

tissue appears, and for a time there remains but the dust of hematic fragments. This, too, disappears, and it becomes finally impossible to recognize the site of the original hemorrhage. Though occasionally a hemorrhage may be absorbed in several weeks, the average is usually three to four months.

In other cases permanent cicatricial changes more or less profound mark the area of a former hemorrhage. These changes are due to the compression of the retina by the escaped blood. The pigmentation that characterizes these lesions has been for the most part derived from the blood pigment rather than from the retina.

Sometimes white or yellowish spots mark the site of a previous hemorrhage. These have been explained as due to fibrin, or to granular or fatty degeneration of the cells. As these spots are most often seen in toxic states, such as nephritis and diabetes, their presence may be due to the local toxic action of the hemorrhage. These spots may disappear in time, but most often persist indefinitely. (Schweigger[2].)

The Causes of Retinal Hemorrhage.—The underlying basis of a retinal hemorrhage is generally a vascular lesion. The hemorrhage may be the first manifestation of a local or systemic sclerosis. Chief among the factors that predispose to hemorrhage is arterial hyperten-

sion. Hemorrhages in the retina, like those in the brain, occur not only in cases of permanent hypertension, but in attacks of transitory hypertension as well.

Venous stasis adds to the danger of hypertension. For this reason both retinal and cerebral hemorrhages occur more often by night than by day. The horizontal position may lower somewhat the systolic pressure, but it makes for venous stasis and vaso-dilatation.

Local vasodilatation plays an important role in hemorrhage. When the arterioles are contracted the venous pressure is low. The venous pulse, then, if it does not exist spontaneously, cannot be induced by ocular compression. If present, it is obliterated by the slightest pressure on the globe. With this low venous pressure, in spite of hypertension, there is little tendency to hemorrhage. On the other hand, when the arterioles are dilated the venous pressure is high. If the venous pulse is present, slight ocular compression will cause an exaggeration of its amplitude. If absent, rather strong pressures on the globe is required to induce its appearance. With this high venous pressure, the retina is particularly exposed to hemorrhage.

The viscosity of the blood may influence also the production of hemorrhage. States Onfray[8]: "The viscosity of the blood creates a re-

sistance antagonistic to arterial tension. To conquer this resistance the tension must rise, and it is possible that in certain cases hypertension is provoked by hyperviscosity. With the arterial pressure unchanged, hemorrhage may be determined by a lessening of viscosity.'' The viscosity of the blood as measured by the viscomieter of Walter Hess ranges normally from 3.8 to 4.5. In pathological states it may be as low as 1.9 and as high as 7.8. Onfray found the index of blood viscosity lowered in numerous cases of retinal hemorrhage.

Abnormal blood states, such as occur in pernicious anemia, leukemia, scurvy, purpura, and hemophilia, predispose in general to hemorrhage. In the latter two affections, however, the retina is rarely attacked.

Pereya[17] calls attention to the frequency of fetinal hemorrhages in malaria. He finds the hemorrhages located near the veins about the disc or near the macula. He considers that alterations in the blood and the malarial toxins produce changes in the vessel wall that favor diapedesis.

Clinically retinal hemorrhages may be considered in the following groups:

1. **Hemorrhage of Traumatic Origin.**—An individual receives a blow on the eye and complains of visual difficulty. The fundus shows one or more hemorrhages, sometimes located in

the center, but more often in the periphery of the retina. Commotio retinæ may or may not be associated. The prognosis fortunately is good, as these hemorrhages are absorbed rather quickly and leave no retinal lesion behind.

Severe compression of the thorax, as may occur on being run over bya vehicle[4, 5], or in railroad accidents[6] has produced hemorrhages in one or both retinæ. The hemorrhages are a result of the venous congestion. The prognosis is generally good.

Another type of traumatic hemorrhage occurs in the newborn. Sicherer and Stumpf[7] in an examination of 200 new-born infants found 42 cases of hemorrhage in the retina and optic nerve. These hemorrhages are most notable early and are generally absorbed at the end of the first week.

2. **Retinal Hemorrhages Due to Abnormalities of the Blood.**—In the diseases characterized by abnormal blood states the diagnosis is usually made by more dominant symptoms than the retinal hemorrhages, which consequently are but of incidental interest in these maladies. The retinal hemorrhages occur most often in leukemia. Caiotti[8] states that in myeloid leukemia the hemorrhages are generally in the anterior segment, and in the papillo-macular region. The vessels are extremely dilated by the enormous increase in white cells; the small-

est ones give way, with consequent hemorrhage.

In pernicious anemia the retinal hemorrhages may be quite abundant, but the vision is relatively little troubled. The hemorrhages are mostly in the posterior segment and are somewhat violaceous. They are due to the hydremia of the blood and fatty degeneration of the vessel walls.

3. **Hemorrhage of Unusual Origin.**—Morax[9] noted a para-macular hemorrhage within 24 hours after an anti-typhoid inoculation. The patient had had a severe general reaction and had complained of some visual disturbance. In a few weeks he completely recovered.

Stock[10] treated a case of miliary tuberculosis in a man of 37 with a fairly strong injection of tuberculin. Some retinal hemorrhages followed. This case had tubercles in the choroid (confirmed by autopsy) and the hemorrhages resulted from the violent local reaction that occurs in the neighborhood of tuberculous foci from injections of tuberculin.

In strong myopes, small retinal hemorrhages may occur, most often in the macular region. But unless the hemorrhage actually involves the macula the lesion is generally too small to cause any visual disturbance. The basis for the hemorrhage is probably capillary injury produced by the stretching of the retina.

4. **The Retinal Hemorrhages of Adolescents.**—An obscure type of retinal hemorrhage affects very occasionally young subjects—from 18 to 30—and males particularly. The onset is rather sudden. The vision in one eye becomes rapidly less and less; seldom are both eyes affected at the same time. There may develop a scotoma in the visual field; the more disturbing, the nearer it is to the point of fixation. If the hemorrhage has broken into the vitreous a thick fog seems to envelop everything.

The hemorrhages are always fairly large, most often peripheral, and more or less numerous. Frequently they are of the pre-retinal type. The veins show inequalities of caliber, sinuosities, darkened color, and a veil of edema. The hemorrhage is hence probably of venous origin. The local arterial pressure is either normal or slightly diminished.

When hemorrhage into the vitreous occurs in this condition it is absorbed rather rapidly. After a few days the ophthalmoscope reveals the blackish mass to be resolved into large flakes, which gradually break up and disappear, leaving the vitreous almost normally transparent. The retinal hemorrhages are likewise absorbed, though sometimes a patch of retinal atrophy results.

The malady is prone to recur—sometimes three attacks come in one year. From repeated

attacks retinitis proliferans often results. Each recurrence leaves behind it an increase of the vitreous and retinal haze, due undoubtedly to the organization of membranes, which in retinitis proliferans is seen in its fullest development.

The etiology of the disorder is not known. The Wassermann test is usually negative, and anti-syphilitic treatment does not prevent recurrences. Tuberculosis of the vessels is quite possibly a factor as well as the lesions of focal infection.

5. **Hemorrhagic Retinitis.**—The conditions that come under this caption will be considered in detail in Chapter XVI.

6. **Hemorrhage Following the Obliteration of Retinal Vessels.**—Hemorrhages due to these causes are not infrequently seen, but the fundus pictures are quite distinctive.

7. **"Essential" Hemorrhages of the Retina.**—Middle-aged individuals who complain of recent visual difficulty often show minute retinal hemorrhages, either single or scattered. These are cases of arterial hypertension, and locally there may be either vaso-constriction or vaso-dilatation. The blood-pressure of the central artery is often only moderately elevated: Diastolic, 50; systolic, 120; the systemic pressure (brachial artery) averages 105/200. The minute hemorrhages are produced by lesions

in the capillary bed. Syphilis is very often the etiologic factor[18].

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CHAPTER XIV

RETINAL EDEMA AND EXUDATES

The various factors that occasion retinal homorrhage may all first produce edema. The exudation of the liquid constituents of the blood occurs, of course, more easily than the loss of the whole blood.

Edema Based on Local Causes.—Traumatic retinal edema "commotio retinæ" may follow a severe blow on the eye. The retina becomes more or less clouded, either in a sector or over its entire extent. The vessels appear darker than ordinarily, but the vessel reflexes are more marked. The visual disturbance is but transient, as in a few days the retina returns to its normal state.

Though immediately following the shock of the accident the vessels are constricted, a little later they become intensely dilated. In this condition serum exudes all the more readily as the intra-ocular tension is usually lowered by the trauma. If the exudation occurs in the nerve-fiber layer the retinal clouding is more marked and more persistent than when the

edema is more superficial. Should edema and hemorrhage arise from the choroid, a traumatic detachment of the retina may result.

The occurrence of retinal edema secondary to vascular obliteration has been already mentioned. In obliteration of the central artery or one of its branches the retinal clouding may represent a nutritive disturbance of the superficial layers rather than a true edema. But in obliteration of a vein there is no question of an actual retinal edema occurring. To a large degree the extent of this edema will depend on local toxic and inflammatory factors.

Edema Based on General Causes.—The principal systemic cause determining retinal edema is nephritis. In renal insufficiency retained salt is deposited in the tissues. As this increases their osmotic tension, the tissues imbibe water. Retinal edema occurs at the same time as edema elsewhere, though the visual disturbance may at times be but slight. The disc, however, will be somewhat elevated, and the outline rather indistinct; the retina may be a bit clouded.

When the edema contains fibrin as well as serum, the term exudate is applied. In albuminuric retinitis the exudate occurs either in large white patches or more or less diffused. Though generally the exudates are accompanied by hemorrhages, in some cases—at least in the early stages—there is but a veil-like

cloud over the retina, affecting particularly the posterior pole[1].

In the typical picture there is first a diffuse clouding about the disc, extending for about two or three disc diameters, and reaching or passing the macula. Quite near the disc are white plaques of albuminuric retinitis and in the region of the macula, the macular star. The serum of the exudate has been rapidly absorbed, leaving behind the deposit of fibrin to which these appearances are due.

The macular star is not at all characteristic of albuminuric retinitis. It may appear in any case of retinal edema. The stellar configuration is merely due to the disposition imposed on intra-retinal exudates by the radial architecture of the perifoveal region.

Why is the fibrinous element so dominant in the edema of albuminuric retinitis? Edema even more than hemorrhage of the retina has a natural tendency to be absorbed after the disappearance of the inciting cause. In the traumatic edema of commotio retinæ the absorption is rapid and total; but in this instance the retinal cells have been bathed in an innocuous medium. In nephritis, however, it is probable that the exudation contains irritant or toxic principles, such as urea and other wastes. These substances may either act as local vasodilating agents, and by this means aggravate

the edema and hemorrhage; or the retinal elements may be directly injured by their contact.

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CHAPTER XV

THE RETINAL CIRCULATION IN GLAUCOMA

In the early stages of chronic glaucomathe retinal vessels show no changes except for the bends made at the border of the disc excavation. In the later stages it is seldom that the vessels maintain a normal appearance. The vessels become dull and without reflections, and it may be difficult to distinguish the arteries from the veins. The arteries often present also various stages of obliterating arteritis. There may be a dilatation of the fine vessels at the base of the excavation, which is indicative of a disturbance in the return circulation. A spontaneous arterial pulse may be noted.

The Arterial Circulation in Glaucoma.—If the local *systolic* arterial pressure were surpassed by the intra-ocular tension, the central artery would be obliterated, and blindness ensue. This, however, rarely happens. But even when the local *diastolic* arterial pressure is surpassed by the intra-ocular tension a considerable disturbance in the retinal circulation is

produced. A jerky blood-flow in the capillaries is substituted for the usual even current. A spontaneous arterial pulse then exists.

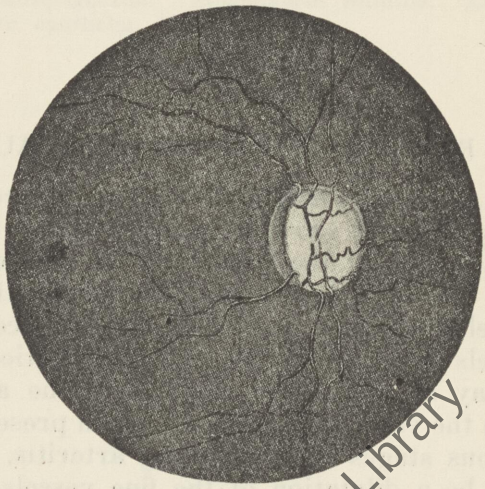

Fig. 38. Old chronic glaucoma. Excavation of the disc. Independently of the cord-like condition of the vessels, note the vascular lacing at the nasal border of the disc, characteristic of trouble with the vascular return.

This condition occurs more often in acute than in chronic glaucoma. Rydel noted a spontaneous arterial pulse in one out of seven cases of chronic glaucoma. Among 57 cases of glaucoma whose circulatory conditions I have studied, 11 showed spontaneous arterial pulsation. Seven of these 11 cases were affected with acute glaucoma. The proportion in favor

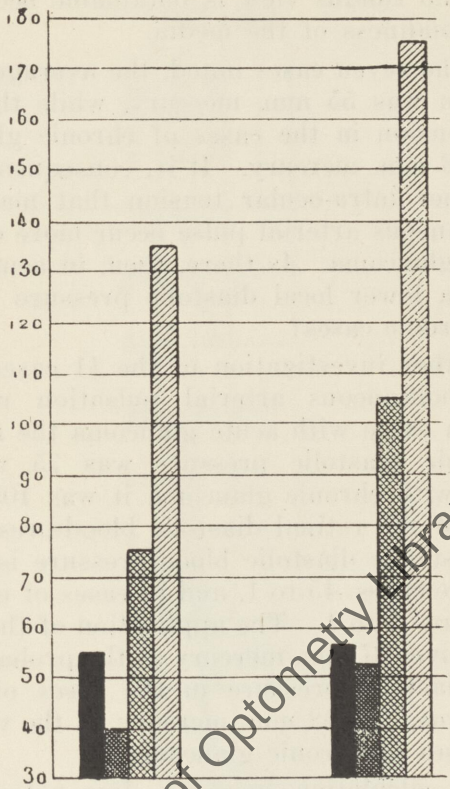

Fig. 39. Showing the different pressures in the author's cases of spontaneous arterial pulse. Left, acute glaucoma. Right, chronic glaucoma.

of acute glaucoma would be even higher were it not that in over half the cases of acute glau-

coma no fundus view is obtainable because of the cloudiness of the media.

In the seven cases noted, the average ocular tension was 55 mm. mercury, while the average tension in the cases of chronic glaucoma was 56 mm. mercury. It is, consequently, not a higher intra-ocular tension that makes the spontaneous arterial pulse occur more often in acute glaucoma. Is there, then, in acute glaucoma a lower local diastolic pressure than in the chronic cases?

Further investigation of the 11 cases showing spontaneous arterial pulsation revealed that in those with acute glaucoma the average systemic diastolic pressure was 75, while in those with chronic glaucoma it was 105. The ratio of the retinal diastolic blood-pressure to the systemic diastolic blood-pressure is in the average cases .45 to 1, and in cases of elevator pressure, .5 to 1. The application of the latter ratio gives 37 mm. mercury as the probable retinal diastolic pressure in the cases of acute glaucoma, and 52 mm. mercury as the value in the cases of chronic glaucoma.

This calculation, however, does not end our study. The question arises as to how the local arterial pressure is influenced by the increased intraocular tension. To decide this point, the blood-pressure in the central arteries was measured in cases where the intra-ocular ten-

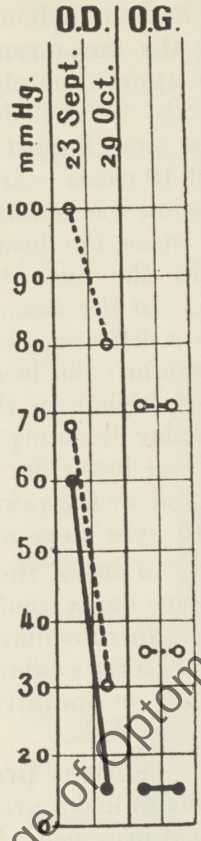

Fig. 40. Fall of local arterial pressure (dotted line) following fall of ocular tension (full line). Chronic glaucoma; sclero-iridectomy.

sion of the two eyes was different. If intra-ocular tension had no influence on the local blood-pressure, the measurements in the two eyes should be approximately the same. We limited our study to the diastolic pressure, which is both the more important and the easier to measure. Of 19 cases examined, in four the intra-ocular tension was about the same in both eyes; in these cases the local blood pressure measurements in the two eyes were likewise about the same. In the remaining 15 cases in which there was a difference of tension between the two eyes, in nine the local blood-pressure was considerably higher in the eye with the greater intra-ocular tension; in two the local blood-pressure was lower in the eye with the higher tension, and in the remainder the measurements in both eyes were about the same.

In the majority of cases, then, the local diastolic blood-pressure has a tendency to rise with the increase in intra-ocular tension. Conversely, when the intra-ocular tension is lowered by pilocarpin or operative measures, the local blood-pressure falls.

Owing to the conditions prevailing in glaucoma, it is difficult to make precise study of the local systolic blood-pressure. Like the diastolic pressure, it rises with the intra-ocular tension, but to a relatively less extent.

The Venous Circulation in Glaucoma.—

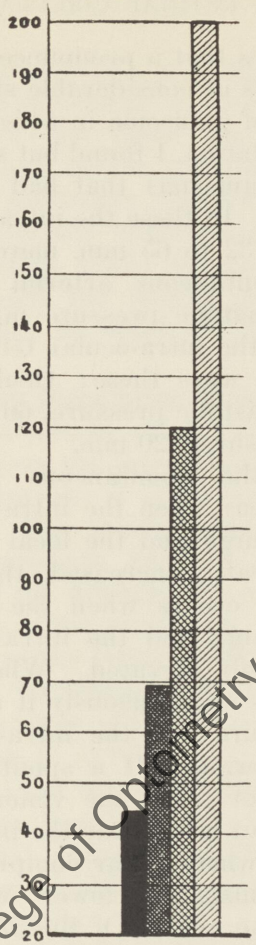

Fig. 41. The different pressures in the author's cases of spontaneous venous pulse. From left to right: Ocular tension, minimum retinal arterial pressure, diastolic arterial pressure, and systolic arterial pressure, the last two taken at the brachial artery.

Elliot[1] believes that a pronounced venous pulse in glaucoma is of considerable significance. In the 57 cases of glaucoma in which I studied the retainal circulation, I found but seven (all cases of chronic glaucoma) that had a spontaneous venous pulse. In these the intra-ocular tension ranged from 32 to 65 mm. mercury, and none showed a spontaneous arterial pulse. Hence the local diastolic pressure must have been greater than the intra-ocular tension. The average figures were these: Ocular tension, 42 mm.; local diastolic pressure, 66 mm.; systemic diastolic pressure, 120 mm.

Whereas the spontaneous arterial pulse tends to appear when the intra-ocular tension is relatively high and the local diastolic pressure not greatly increased, the spontaneous venous pulse occurs when the local diastolic pressure is high and the intra-ocular tension only moderately elevated. When the venous pulse appears spontaneously it means that the venous pressure and the intra-ocular tension are in equilibrium. If a spontaneous venous pulse does not exist, the venous pressure is either higher or lower than the intra-ocular tension; higher when ocular compression induces the venous pulse, and lower when this procedure is of no avail. Of the 57 cases of glaucoma studied, in three was the venous pressure higher than the intra-ocular tension, and in 47 it was lower.

The venous circulation goes on, nevertheless, because of the resistance of the venous walls, the impulse of the blood current, and the transmitted force of the intra-ocular tension. But were the difference between venous pressure and intra-ocular tension high enough, the venous circulation would be checked. This I have never noted, but then it is only possible to examine cases in which the media are still transparent.

The Capillary Circulation in Glaucoma.—Let us consider the case in which the local diastolic pressure is above the intra-ocular tension, and the venous pressure is equal or only slightly inferior to it. The average capillary pressure will be intermediate between these values; higher toward the arterial end of the capillary bed, and lower in the periphery. It would happen then that in the latter portion of the capillary bed the circulation may be arrested in diastole by the force of the intra-ocular tension, and only go on in systole.

Where the local diastolic pressure is below the intra-ocular tension, there will be a spontaneous arterial pulse. The arterioles themselves will now be closed during diastole, and only open in systole.

These circulatory disturbances explain various functional disturbances that occur in glaucoma, such as the contraction of the visual field

and the appearance of scotomata.

Vascular Spasms in Glaucoma.—Besides a progressive diminution of vision, glaucomatous patients are subject to attacks of obscuration. For several minutes or several hours the objects will appear as if seen through a veil or fog.[2]

These periods of obscuration are due to attacks of spasm affecting the retinal vessels. They occur most often in eyes showing the lesions of obliterating arteritis.

The Mechanism of the Retinal Circulation in Glaucoma.—The most dominant fact in the retinal circulation in glaucoma is the increased venous pressure. In the normal eye the venous pressure ranges from 18-20 mm. mercury; in glaucoma it often reaches 60-70 mm. mercury, sometimes surpassing the increased ocular tension. Ophthalmoscopically a dilatation of the venous trunk can be noted as soon as the latter enters the eyeball. This dilatation is also noticeable in the anterior ciliary veins as they leave the globe to pass under the conjunctiva.

The high venous pressure is only partly due to an increase in the local arterial pressure. In subjects with marked local hypertension, but not affected by glaucoma, the venous pressure is little above the normal figure. It is then the heightened intra-ocular tension that creates the venous hypertension. When the arm is con-

stricted to a degree below the brachial systolic pressure the circulation therein still goes on. An elevation of venous pressure overcomes the resistance. In the eye a similar phenomenon occurs, and as the intra-ocular tension rises, so does the venous pressure.

For a given increase in intra-ocular tension, the circulatory disturbance is the greater, the less the systemic (and by consequence the local) arterial pressure. Thus acute glaucoma, which is seldom accompanied by hypertension, shows the most severe ocular disturbance. Chronic glaucoma, on the other hand, tends to occur more often in hypertension cases. Sulzer[3] maintained that a heightened intra-ocular tension by itself would not suffice to produce the symptoms of glaucoma, if the arterial pressure were sufficiently elevated so that the circulation would not be appreciably checked by the exaggerated tension.

To the circulatory insufficiency is due the degeneration of the optic nerve-fibers and the resulting glaucomatous excavation, rather than directly to the increased tension. In chronic arterial obliteration, the excavation of the optic disk results without the accompaniment of any elevation in intra-ocular tension, and is due solely to the ischemia of nerve and retina.[4] The circulatory disturbance in these cases is evident. The vessels are constricted, the local

arterial pressure is sometimes quite low, and the local systolic pressure is always nearer than normally to the diastolic finding. The subjective symptoms are often similar to those in true glaucoma.[5] The two conditions are similar and so in this respect, in both, the pressure relations between the retinal circulation and the intra-ocular tension has been disturbed.

The choroidal venous pressure in glaucoma becomes elevated with the retinal venous pressure, as is evidenced by the dilatation of the anteriorciliary veins.

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CHAPTER XVI

THE RETINAL CIRCULATION IN HEMORRHAGIC RETINITIS

Albuminuric Retinitis.—In the early stages of albuminuric retinitis the vessels are engorged with blood, the veins are dilated and tortuous, and the arteries display brilliant reflexes. Later, when the retina thickens and becomes cloudy, the arteries appear hazy, as if buried in the papillary tissue and the clouded retina. The hemorrhages and edema are localized near the papilla, covering an area of 5-8 mm. about the disc. Should one find in any case the hemorrhages disseminated to the limits of the ophthalmoscopic field, the condition must be considered as a local vascular affection rather than characteristic of albuminuric retinitis.

In the last stages the hemorrhages increase, and the appearance of the vessels becomes abnormal, often in part they seem replaced by white cords, particularly the arteries. The veins are narrowed and pale, due to the poverty of the retinal circulation.

The local arterial pressure is considerably elevated. This symptom occurs early and can be noted while the visual function is still intact and the ophthalmoscopic examination otherwise negative. Certain subjective symptoms may accompany this early stage, such as attacks of visual obscuration, specks before the eyes, and a sensation of haze. A retinitis without local arterial hypertension is probably not on a nephritic basis.

The venous pressure becomes elevated also, but only to a very moderate extent and not nearly as early as the rise in the local arterial pressure. With this venous hypertension there is associated a local vasodilatation.

Greef in 1903 maintained that the same cause which occasions the lesions of the renal vessels affects likewise those of the retina, and that disease of the latter vessels may precede that of the former. According to this theory, an arterio-capillary fibrosis affects all the organs, but particularly those of the kidney and the retina. Weiss[1] claims to have noted by capillaroscopy a disturbance of the capillary circulation in nephritis.

Rochon-Duvigneaud finds no support for this theory in histopathological study. He believes that the lesions of nephritis are not due to vascular changes, but are primarily determined by the insufficiency of renal elimination. The cir-

culatory disturbances in the retina, though occurring early, are but a secondary manifestation.

The lesions in the retina are not due simply to hypertension, for in hypertension not due to nephritis there is not the picture of albuminuric retinitis. May it not be that the nitrogen excess in the blood, besides exercising a general vaso-constrictive action by its effect on the nerve centers, acts on the peripheral vessels, especially those of the kidney and retina, as in vasodilator?[2]

The findings in the retinitis of pregnancy are very similar to that in albuminuric retinitis.[3]

Diabetic Retinitis.—In many cases of diabetic retinitis renal insufficiency may be a factor as well as diabetes in the production of the pathology noted. Many of these patients show albumen as well as sugar in the urine.[4]

In diabetic retinitis the vessels are generally less dilated than in albuminuric retinitis, the arteries and veins are less brilliant or even dull, and the local hypertension, if present, is much less marked. As the vascular lesions are not constant, the retinal hemorrhages and edema that occur are probaly due to paroxysmal attacks of hypertension, brought on possibly by exertion or dietetic indiscretions.

Leukemic Retinitis.—The fundus in leukemia

is very light colored, as are likewise the retinal vessels. Grunnert[5] noted that the circulation in the large veins was often suggestive of the flow of sand in an hour-glass. This granular appearance, if not present spontaneously, can be induced by pressure on the globe strong enough to overcome the diastolic pressure of the central artery. This granular appearance is due to the enormous number of white cells in the circulating stream. It is this that is mechanically responsible for the disturbed retinal circulation, and for the vascular lesions that occur in this condition.

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CHAPTER XVII

THE RETINAL CIRCULATION IN DISEASES OF HEART VESSELS AND BLOOD

I have had the good fortune, thanks to the courtesy of Professor Vaquez, to examine in his clinic a great number of patients suffering from cardiac and vascular disease. My findings indicate that a routine fundus examination in this class of cases, if adopted by the internist, would be of considerable value to him.

Diseases of the Heart.—A study of the retinal circulation does not generally give any special diagnostic help in cardiac affections. However, the spontaneous arterial pulse of aortic insufficiency may be noted in patients who considered themselves quite healthy. Even when this spontaneous pulse does not appear, there is generally an abnormal vibration of the arterial walls.

The spontaneous venous pulse becomes somewhat more pronounced when a distinct jugular pulse is present. An alternation of the pro-

voked venous and arterial pulses is most often seen in diseases of the right heart, but this "doubled retinal pulse" is encountered in other conditions also.

The aspect of the retinal vessels in heart disease is not appreciably modified till the terminal stages. As soon as the asystole of cardiac exhaustion sets in, the retinal circulation becomes markedly affected. The arteries and veins darken and can hardly be distinguished from each other. Ocular compression provokes but enfeebled pulsations that are easily extinguished.

Diseases of the Vessels.—The hypertension of the retinal vessels that accompanies systemic hypertension has already been considered in detail, as well as the spasm of the retinal vessels that so often is associated. Occasionally I have noted in hypertension cases that complained of no visual difficulty, small retinal hemorrhages. These have disappeared with the passage of time, leaving behind no traces.

Outside of these hemorrhages I have never seen ophthalmoscopic signs of arterial or venous lesions without the patient complaining of visual disturbance. Capillary sclerosis, however, is often met in patients beyond the fifth decade. This condition is indicated by a more or less pronounced anemia of the retina, con-

tracted arteries, and a diminished venous pressure.

Diseases of the Blood.—In pernicious anemia the contracted pale arteries are in contrast to the dilated veins. Retinal hemorrhages are

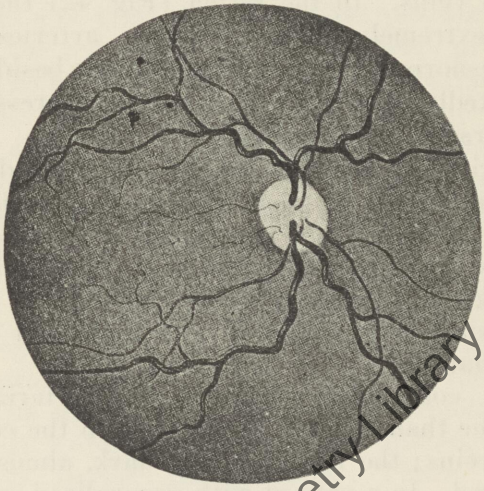

Fig. 42. Appearance of the retinal vessels in erythemia. (Vaquez' disease.) The vessels are tortuous and remarkably augmented.

constantly noted—even in patients who complain of no visual disturbance.

In the other forms of anemia the lesions are less characteristic. Generally the fundus is pale, the venous pressure quite low, and the ocular tension diminished.

In erythremia there is a distinctive fundus

picture.[1] This disease is characterized by a large increase of red blood cells, a ruddiness of the skin (especially that of the face and extremities), and a hypertrophy of the spleen. The conjunctiva shows a network of dark and dilated veins. In the retina (Fig. 42) the veins are extremely dark, while the arteries have their normal color; and the veins besides are markedly sinuous. The systemic pressure is generally normal, but that of the retinal arteries is usually slightly augmented. The visual acuity is good, though very often the affected individual complains of transient attacks of obscuration, and a sensation of black specks or sparkling points before the eyes—the subjective evidence of a disturbed retinal circulation.

In congenital cyanosis, the arteries are darker than normal, and approach the color of the veins; the retina is also dark, almost slate colored. In a patient with generalized cyanosis due to stenosis of the pulmonary artery, I noted a similar fundus. Occasionally retinal cyanosis may be noted before the general cyanosis has appeared. In a case of contraction of the pulmonary artery reported by Babinski and Roufesco,[2] the skin was of normal color, the nail matrices displayed a slight violaceous tint, but the retinae presented a distinct cyanosis.

The following case of Baraquis'[3] deserves mention. A child of 11 years, afflicted with typical congenital cyanosis, suddenly experienced diminished vision, after a spell of dyspepsia accompanied by dyspnea. The sight of the right eye was limited to light perception; that of the left eye to hand movements at one foot. The lids and conjunctivae were markedly cyanotic. The pupillary reactions to light were feeble; there was exopthalmos and moderate mydriasis. The irides, blue in color when the child first entered the hospital, became chestnut-brown. The change in color was due to the extreme venous congestion of the iridic vessels, as at death the irides returned immediately to their blue color. An ophthalmoscopic examination was only possible in the left eye. The arteries could not be distinguished. The veins were dark, sinuous, and dilated. The disc was hardly recognizable. There were hemorrhages in the macular region, and between the disc and the equator. Autopsy demonstrated a hypoplasia of the pulmonary artery, with adhesions between its valves, and a persistence of the foramen of Botal. The retinal arteries were extremely small; and the large veins of the retina showed a proliferating endophlebitis.

In cyanosis of the retina compatible with normal vision, the arteries and veins are both

dilated. The bulbar vaso-constrictor center is only moderately stimulated by the accumulated carbon dioxide of the blood, so that the visceral vessels are only affected. In grave cases, however, there is a generalized contraction of all the arteries, with later hypertrophy of the muscular coat and endothelial changes.

In every case in which cyanosis has affected the face, cyanosis has also been noticeable in the retina.

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CHAPTER XVIII

THE RETINAL CIRCULATION IN CEREBRAL DISEASE

The retinal circulation often furnishes useful information concerning the state of the cerebral circulation. Just as arteritis may be localized to a single artery or affect the entire retina, so it may continue further and extend to a greater or less degree into the cerebral circulation. In any case we can surely learn more from a direct observation of the retinal vessels and the determination of their blood-pressure, than is possible from palpation and auscultation of the radial or brachial arteries.

Rahlmann[1] in 1889 made a study of concomitant vascular lesions of the brain and retina. He concluded that sclerosis of the retinal vessels generally connotes sclerosis of the cerebral vessels. Hertel,[2] basing his opinion on autopsy findings, considered that definite arteriosclerosis of the retinal vessels indicated a similar state in the cerebral circulation, but that a negative finding was without significance.

Many observers have noted clinically that

vascular lesions in the brain frequently follow vascular lesions of the retina.[3, 4] In my experience, cerebral lesions are more likely to be associated with the occurence of hemorrhages in the retina rather than with simple sclerosis of the retinal vessels. Noting the former condition (albuminuric retinitis excepted) the ophthalmologist may justly fear that the same danger menaces the brain. The hemorrhages resulting from arterial hypertension carries grave possibilities, as also does venous thrombosis. Many cases, however, have survived for years after the advent of hemorrhagic retinitis, without the appearance of the dreaded cerebral disturbances—mental troubles, softening, hemiplegia, or death. Foster Moore, in a study of 62 cases, found the average length of life after the development of venous thrombosis was 5-8 years.

The Retinal Circulation in Choked Disc

Characteristic of choked disc, besides the edematous swelling of the optic nerve, retinal edema, and occasional peripapillary hemorrhages, are certain changes in the appearance of the retinal vessels. The arteries become extremely narrowed, sometimes hardly distinguishable. Buried in the papillary edema they can often only be recognized through the pulsation induced by ocular compression. The veins

are slightly dark, somewhat dilated, and markedly sinuous.

The local arterial pressure in choked disc, whatever be the cause, is usually elevated,

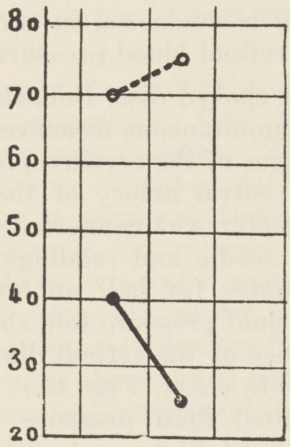

Fig. 43. Fall of cerebro-spinal pressure. Full line shows cerebro-spinal tension; dotted line shows diastolic retinal arterial pressure; both in mm. Hg.

though the systemic pressure may remain normal. This rise in local pressure is sometimes very slight, and sometimes absent. The venous pressure is generally normal.

After decompression produced either by lumbar puncture or craniotomy the local arterial pressure generally falls. To a certain point the blood-pressure in the retinal arteries parallels

the pressure in the cerebrospinal fluid. With an increase in the pressure of the cerebrospinal fluid, there is an increase in the pressure of the cerebral arteries—otherwise anemia of the brain would ensue. The increase in cerebral blood-pressure is evidenced in turn by an increase in the retinal blood-pressure.

In a case of choked disc, Bollack and I were able to make simultaneous measurements of the pressure changes of the cerebrospinal fluid and those of the central artery of the retina. A Claude manometer was connected with a lumbar puncture needle and readings were taken every ten minutes for half an hour. As the cerebrospinal fluid pressure fell, the immediate effect was a rise of the retinal diastolic blood-pressure. (Note chart, Fig. 43.) The fall of the cerebrospinal fluid pressure allowed the cerebral vessels to dilate, and augmented their flow of blood, with a consequent rise in the local blood-pressure. (This explains how cerebral hemorrhage may follow simple lumbar puncture.) After this primary dilatation of the cerebral arteries, the cerebral circulation probably becomes regulated to the fall in cerebrospinal fluid pressure, with a subsequent reduction in the blood-pressure of the cerebral, and consequently of the retinal arteries.

In a histo-pathological study of choked disc, Dupuy-Dutemps observed that the central

artery was not at all contracted in the intraneural part of its course; but that the central vein was already considerably flattened in the pial sheath. He concluded, therefore, that the papilledema was due to the compression of the ophthalmic vein in its passage across the intervaginal sheath.

This theory of the mechanism of choked disc follows that proposed by Deyl, but is not supported by our observations. The ophthalmoscopic appearance of choked disc is quite different from that of venous obstruction. Moreover there is no rise in the pressure of the central vein as would be expected if more than a minor interference with the return circulation occurred. This theory likewise fails to account for the relations above noted between the pressure in the central artery and that of the cerebrospinal fluid.

The Retinal Circulation in Intracranial Hypertension Without Choked Disc.—In some cases of intracranial hypertension, particularly in serious meningitis, there may be no change in the appearance of the optic disc. These cases, nevertheless, will generally show a considerable rise in the retinal diastolic blood-pressure. This diastolic hypertension is relatively more pronounced than in choked disc, and furnishes a very important diagnostic aid, when the ophthalmoscopic signs otherwise may

be negative. Local hypertension always can be diagnosed when the diastolic pressure of the retinal artery is over one-half that of the brachial artery.

I have been able to clinically demonstrate the relation of intracranial hytertension to the pressure of the retinal artery. A child, following skull fracture, had a large resection of the frontal bone, which permitted one to exert direct compression on the brain. Such compression immediately produced an elevation of the retinal diastolic pressure, which disappeared with its cessation.

The Retinal Circulation in Epilepsy.—In the epileptic seizure the extreme pallor of the face is accompanied by an equally extreme anemia of the retina; the retinal arteries become almost effaced. This is followed by a more or less transient hyperemia, lasting from one to two hours in a mild spell, up to twelve hours after a violent attack.[5, 6] The retina and disc remain pale, but the veins become dilated and sinuous.

I have examined many epileptics between seizures and have failed to note then any anomalous condition of the retinal circulation. However, in the following case I found a disturbance of function:

The patient, age 39, was buried by a shell explosion in August, 1917, and thereafter suf-

fered amnesia for three months, and experienced convulsive attacks monthly. The attack lasted from 15-25 minutes, and was preceded by an aura of the ringing of bells, but no visual disturbance. After the attack a sensation of sudden blindness supervened for 5-10 minutes; one time it continued for two hours. In an examination during an aura, the systemic blood-pressure was somewhat raised: 150/100; but the retinal blood-pressure was relatively low: 60/30.

The Retinal Circulation After Skull Injuries.—Among the numerous subjective ills experienced by the victims of cranio-cerebral injuries, those in relation to vision are most frequent. There is generally a sensation of dazzling, and the sudden appearance of mist before the eyes—lasting for several minutes and aggravated by brusk body movements. Accommodative asthenopia often accompanies this condition, with rapid fatigue on reading, and on visual application of any type; occasionally there are attacks of transitory amaurosis.[7]

A personal examination of a great number of skull injury cases at the service at Val-de-Grace showed that these subjective disturbances may often exist in patients whose fundi are ophthalmoscopically normal. The retinal blood-pressure, however, is generally in these cases either higher or lower than the norm. An ex-

acerbation of the headache and of the subjective visual disturbances is often accompanied by a rise in the retinal arterial pressure. On the other hand, it is interesting to note that in albuminuric retinitis, which is characterized by a notably high local arterial pressure, lumbar puncture by lowering this may produce a distinct amelioration of the visual condition.

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CHAPTER XIX

RETINAL HYPEREMIA AND ISCHEMIA

When there is a diffeernce in the appearance of the disc and the vessels in the two retinae, hyperemia is easy to distinguish, but when the appearance of the disc and vessels on both sides is the same, it often becomes quite difficult to make a definite statement.

Retinal hyperemia may be produced by vasomotor disturbance. In animal experimentation, this is accomplished by section of the cervical sympathetic. In man, dilatation of the arteries and veins is produced by the inhalation of amyl nitrite. With the increased activity of the retinal circulation, the color of the veins approaches that of the arteries. The pressure becomes raised in the central vein, and on ocular compression the phenomenon of an alternate arterial and venous pulse can be produced. This "double retinal pulse" is characteristic of local vasodilatation and active hyperemia.

Clinically, retinal hyperemia may follow exposure to strong light, corneal and conjunctival irritation, and accommodative asthenopia. It

can be noted also in the earliest stages of arterial hypertension and other disorders, such as albuminuric retinitis and optic neuritis. The hyperemia may induce a subjective sensation of dazzling, or of spots before the eyes, or sparks in the visual field.

Passive hyperemia is characterized by a darkening, and a sinuous dilatation of the veins, an increased ruddiness of the disc, and a moderate increase of venous pressure. This condition is to be seen in venous thrombosis, erythremia, asystole, emphysema, orbital tumors, arteriovenous aneurysm, etc.

Retinal Anemia.—Pallor of the disc is not by itself a definite indication of retinal anemia. The condition of the vessels must be studied. Retinal anemia is seen most typically in man in obliteration of the central artery. Retinal angiospasm may be associated with an extreme degree of anemia. In arteritis of the retinal vessels the anemia is partial, as the blood supply is diminished in this condition.

In all of these instances the arterioles are narrowed, and ocular compression produces but feeble arterial pulsations. Though the arterial pressure may often be elevated, the venous pressure is always low, as the arterial pressure has then been almost exhausted in its passage through the contracted capillaries. In the aged,

such a condition of the circulation in greater or less degree is generally seen.

Retinal anemia may follow the operation of ligating the common or internal carotid for pulsating exophthalmos. In a case in which both common carotids were ligated,[1] the visual acuity became considerably impaired (R. E. 20/60; L. E. 20/80), and the visual fields markedly contracted. The retinal systolic pressure was very low—30 to 40 mm. mercury. Couchoix[2] found that in 36 cases of double ligation of the common carotids, in 10 the retinal function became seriously affected.

Unaffected

Axillary P.	Carotid P.
120	40
115	45
160	50
180	110

Affected

Axillary P.	Carotid P.
85	15
135	18
180	30

Duvillier[3] has experimentally studied the effects of double ligation of the common carotids. In the dogs who seemed to suffer no cerebral disturbance, the pressure in the carotid artery as compared to that in the auxiliary artery was distinctly much higher than in those

who showed the signs of cerebral changes. This is well illustrated by the following protocol:

Under certain circumstances ischemia may affect not the entire retina, but only a portion, as in angiospasm or arteritis localized to a branch vessel.

The Retinal Anemia in Quinine Amblyopia

The amblyopia from quinine has been attributed by some to retinal anemia; by others, to a toxic action on the ganglion or percipient cells.

Giraud[4] examined a case four days after the ingestion of a massive dose of quinine. The systolic pressure of the brachial artery was then normal, and the retinal arteries showed no visible changes. One month later, however, the arteries displayed a distinct piping of periarteritis.

Bollack's case[5] was observed from the ninth hour of quinine intoxication. The retinal arteries were then moderately contracted, but the next day and thereafter appeared normal; the disc pallor, however, continued. At first when the vision was impaired there was a marked retinal hypertension, which disappeared on the second day with the re-establishment of visual function. In attempting to explain this transitory retinal hypertension, with associated disc pallor and apparently normal vessels, Ballock assumes an intense vasoconstriction of the finest arterioles. This causes the rise of pres-

sure in the arterial trunks at the papilla, and a capillary anemia. This capillary anemia, however, is difficult to perceive beyond the region of the disc.

Extreme degrees of retinal anemia in quinine amblyopia have been noted by many observers, with the arteries and veins extremely thin and almost invisible beyond the disc. Michel noted such a condition one month after quinine poisoning, and in Gruning's case the vessels were still fiiliform three months after. The macular region at times may show a change of color similar to the cherry-red spot in acute obstruction of the central artery.

Retinal anemia appears then to be the constant and dominant feature in quinine amblyopia. Lesions of the ganglion cells and of the optic nerve fibers have been noted in man, and experimentally in animals, but these may be the result of the prolonged circulatory disturbance.[6]

Vail and Valiere-Vialeix[9] have observed that hypertension of the central artery may be present with reduced intra-ocular tension. This condition speaks for vasoconstriction, even though there be no decoloration of the retina.

Schlippe[11] believes that a bilateral optic atrophy which he encountered in a girl of 17 was due to intra-uterine quinine intoxication. The mother had taken, during gestation, large

doses of quinine in the treatment of a severe attack of malarial fever.

Antipyrin intoxication produces a condition similar to that in quinine amblyopia, and probably through a similar mechanism.

Retinal Anemia in Post-hemorrhagic Amaurosis

An amaurosis, which may remain permanently, sometimes affects either one or both eyes after one or more severe hemorrhages. The amaurosis may be partial or total, and appear either immediately after the hemorrhage, or, as more often happens, several days later, but rarely after the sixteenth day.

In most cases, though not all, the ophthalmoscopic examination reveals a very marked anemia of the retina. Is then the early amaurosis and the later consecutive optic atrophy due to this anemia? Terson[7] considers that this factor alone is not adequate, for not only have there been cases without ophthalmoscopic evidence of ischemia, but amaurosis only exceptionally complicated the numerous severe hemorrhages in the late war.

Magitot[8] reported a case of post-hemorrhagic amaurosis following hematemesis. The retinal-arterial pressure was extremely low. In the horizontal position, it measured 40/15, and in the vertical posture was still lower. Magitot believes that the amaurosis can be sufficiently

well explained by the local hypotension and the accompanying anemia.

Retinal anemia when prolonged, whatever be the cause thereof, is productive of grave consequences to the nervous tissue of the retina. Optic atrophy may follow even the chronic vascular lesions that but partially obliterate the retinal vessels.

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CHAPTER XX

THE TREATMENT OF DISORDERS OF THE RETINAL CIRCULATION

Locally very little can be done for disorders of the retinal circulation. The general status of the individual must be investigated and the etiologic factors discovered, after which treatment can be intelligently instituted. The association of internist and ophthalmologist is as indispensable in the treatment as in the diagnosis. The ophthalmologist must actively direct the therapeutic measures, just as he would in a leutic lesion. The importance of the eye is so great that the ophthalmologist must prepare to accept full responsibility in whatever relates to it.

But here, as elsewhere, the oculist must govern himself in accordance with the special circumstances of the case. Called, because of a complicating ocular lesion, to the bedside of the patient with a serious heart or kidney disease, he cannot presume to other than a secondary role. But when the individual complains pri-

marily of some visual disturbance, whether the condition be purely local or the ocular manifestation of a systemic disorder, we should maintain, at least to a certain measure, the direction of his treatment.

Though the treatment of circulatory disorders of the retina must in each case be directed against the special etiologic factors involved, the following general considerations merit attention:

1. Treatment of Retinal Hypertension—the Regimen.—When retinal hypertension is a manifestation of a systemic process, reliance must be placed on general measures, and particularly on control of the patient's regimen. Neither alcoholic beverages nor highly spiced or salted foods can be allowed. The diet should be mainly lacto-vegetarian, the amount of meat restriction depending on the stringency of the case. Salt should be used as little as possible. The fluids should be limited to about 1,500 cc. per day. The excretion of urine must be watched and should be at least 1,000-1,200 cc. in 24 hours. As renal elimination appears to be facilitated in the horizontal position, it may be of advantage to the patient to drink of water before arising and after retiring.

Regulated physical exercise is to be encouraged, but strain and fatigue must be avoided.

As adequate and proper sleep is most essential, late hours are to be rigorously proscribed.

2. Physiotherapy in the Treatment of Retinal Hypertension.—Hydrotherapy has been advised. The cutaneous excitation perhaps relaxes the peripheral vasomotor nerves, and thus lowers the systemic pressure. In any case the treatment can do no harm. The carbonated waters appear to be particularly beneficial.

Vaquez[1] considers the high frequency current to be quite worthless in the treatment of hypertension, and supports his views by the testimony of Broca, Babinski, and Widal.

Radiotherapy of the lumbar region has been tried with the idea of diminishing the activity of the suprarenal glands. The method is still experimental.

3. The medicinal Treatment of Hypertension.—Vasodilating drugs, for the most part belonging to the group of nitrites, are in use for their hypotensive effect, but their action is very brief—two or three hours at most. These drugs act rapidly; in the case of nitroglycerite the effect is manifest in one minute and is at its maximum in five minutes. With amyl nitrite the fall in systemic pressure is at its maximum 15 seconds after inhalation, and is over by the second minute.

The peripheral dilatation thus induced means an increased flow and an increased pressure in

the retinal arterioles, capillaries and venules. Because of this the nitrites are as dangerous in certain cases as they are valuable in others. Suppose a patient with systemic hypertension has retinal hemorrhages, a marked retinal hypertension, and dilated vessels. The administration of vasodilators in this case would but favor further hemorrhages and exudation.

Another case of systemic hypertension may show constricted vessels surrounded by perivascular connective tissue. The character of the arteries and the associated low venous pressure indicate that the process of obliteration has already affected the capillary bed. The nitrites (would that their action was less fugacious!) will here exert a desired and favorable action.

Recently organotherapy has been in considerable vogue, but the results are still *subjudice*.

When the hypertension is based on syphilitic infection, as not infrequently occurs, anti-luetic treatment with the arsenobenzols and mercury must be instituted. Insoluble mercurial preparations, such as gray oil or calomel, or soluble salts are equally efficacious. I have a preference for the intravenous injection of mercuric cyanide. Oral administration is conceded to be unreliable; nevertheless one may resort to it, or, better still, the rectal route, when necessary to avoid alarming the patient. Because albu-

men or sugar is found in the urine the possibility of syphilis should not be dismissed. Even in obscure cases, in which lues cannot be proven, mercurial treatment has often been of value.

The iodides, though used extensively, have but a slightly hypotensive action. They seem particularly indicated in endarteritic lesions. In high doses the iodides tend to diminish the viscosity of the blood, and it is probably by this means that they lower the blood pressure. But this diminished viscosity is favorable to retinal hemorrhages, the possibility of which should be in mind in the use of this drug.

4. The Treatment of Obliterative Vascular Conditions.

(a) Arterial Spasms.—The vasodilators are the chief agents in the symptomatic treatment of arterial spasm. In the use of amyl nitrite the first inhalation at least should be under the observation of the physician. Opiates act well as adjuvants, and in particularly severe cases a hypodermic injection of morphine is indicated. In a subacute case the following prescription is serviceable:

Morphin, or heroin hydrochloride	.06	gr.j
Nitroglycerine (1% alcoholic sol.)	4.	mlxxv
Aquae rosae	20.	zvj
Aquae destilatae	100.	oz. iv.

S. A teaspoonful in coffee two-three times daily.

When the spasms tend to recur antispasmodics of the type of valerian are indicated. A pill made up of five centigrams (¾ grain) each of zinc oxide, extract of hyoscyamus, and extract of valerian can be recommended. This dose should be given one to three times daily for five consecutive days.

(b) Arterial Obliteration.—In arterial obliteration our chief supports are iodides and the vasodilators. Should the case be luetic, these remedies should complement the specific treatment.

Arterial spasms produce attacks of transient blindness. These spasms favor thrombus formation and subsequent arterial obliteration. Consequently the moment an attack of obscuration comes the patient should inhale an ampoule of amyl nitrite. It would be well for those thus afflicted to carry an ampoule of amyl nitrite always with them.

Once arterial obliteration becomes established, be it from thrombus or embolus, immediate attention is imperative. If 24 hours have already elapsed from the advent of blindness, it is almost certain that nothing will be availing, but nevertheless active treatment should be instituted. Amyl nitrite is the main reliance in this emergency, and should be in-

haled every three to four hours. Another aid is prolonged massage of the globe. Paracentesis of the cornea has frequently been of value, the abrupt fall in ocular tension producing some aspiration in the retinal vessels. Furthermore, we can try to force the barrier by increasing the onward pressure of the blood current. Digitalis has been recommended, and it may serve this purpose by briefly exaggerating the action of the left ventricle. It should be among the first measures tried.

(c) Venous Obliteration.—The onset of obliteration in the veins is much slower than in the case of the arteries; the immediate symptoms are not so alarming, and the treatment consequently does not partake of the same emergency character. Our energies should be directed to the determination of the etiologic factor and its control. Syphilis should be thought of, focal infection and acute or chronic disease in the vicinity. If arterial hypertension co-exists, the necessary regimen must be instituted.

Outside of measures thus indicated, certain drugs may be of value, such as urotropine, tincture of hamamelis, or of castanea.

Blood-letting in the Treatment of Vascular Diseases of the Retina.—Blood-letting acts favorably on arterial hypertension, though unfortunately the effect is but transient. Accord-

ingly this measure is useful only in the acute complications of hypertension. Blood-letting may be of particular value in albuminuric retinitis, for besides lowering the blood pressure, this procedure eliminates some of the toxic substances.

Local blood-letting, as with leaches or cupping over the temples or mastoids, enjoyed in the days of our forefathers a great popularity in the treatment of hemorrhagic affections of the retina. Of late their value has been considerably debated. A salutary action could be explained by the anastomoses between the ophthalmic and facial vessels, and, though the practice may not be very efficacious, it certainly does no harm. Pergens[2] maintains that blood-letting in the temporal region is based on a pathologic misconception that has continued for twenty centuries. An entire group of ophthalmic troubles was considered to depend on the penetration into the interior of the eye of liquids derived from the temporal artery or from the temporal region. From this came the idea of making incisions in the temple to drain these fluids before they could effect their entrance into the eye. Cauterization of the artery was done for a similar reason—to close its lumen and so prevent the flow of blood to the eye.

Lumbar Puncture.—In albuminuric retinitis

lumbar puncture sometimes occasions an improvement in symptoms. In all cases of increased cerebrospinal fluid pressure, lumbar puncture will generally cause a disappearance of the paroxysmal attack of visual obscuration that is so disturbing to the patient.

The Treatment of Retinal Anemia.—The treatment of acute retinal anemia is that of acute arterial obstruction. For the retinal anemia that sometimes follows large hemorrhages, A. Darier believes in securing a severe reaction by the use of retro-bulbar injections. For the chronic types of retinal anemia dionin (5%) has been instilled, but a favorable effect has not been demonstrated.

Certain forms of optic atrophy are secondary to retinal anemia. Leriche thought the condition could be remedied by denudation of the internal carotid, thus stripping it of the vaso-constrictor fibers of the retina and producing a dilatation of the retinal vessels. The dire effects of retinal anemia come on too early for this procedure to be of any value. Of what avail is the re-establishment of the retinal circulation when the ganglion cells and the optic nerve fibers are degenerated? In obliteration of the central artery, the blindness persists, even though the retinal circulation becomes rapidly restored.

Abadie[5] has recently advised for the treat-

ment of retinal anemia the injection into the orbit of a sterilized solution containing one milligram of atropin to paralyze the contractile fibers of the arterial tunics. These injections may be repeated from time to time. On the days they are not given, the patient should take 1 mg. of atropin by mouth. Lacta[6] states that he employed this treatment successfully in a case of quinine amaurosis.

The Treatment of Retinal Hemorrhages.—In the treatment of retinal hemorrhages primary attention should be paid to the etiologic factors —be it hypertension or local or systemic vascular disease. For the direct treatment of the hemorrhage, injections of ergotin and adrenalin have been used, but are without value.

The recurrent hemorrhages of adolescents, a disease of unknown etiology, furnishes an example of the inefficacy of merely symptomatic remedies. Some favorable effects have followed the injection of horse serum, or of specially prepared hemostatis serum. Aubineau[4] has used subconjunctival and subcutaneous injections of diphtheria antitoxin, or of plain horse serum. One of five cases appeared to show improvement. Some day when the cause of this malady is discovered, the condition will be under control and recurrences definitely prevented.

The Local Treatment of Vascular Affections

of the Vascular Affections of the Retina.—Miotics appear to be vasoconstrictors, and mydriatics, vasodilators of the ocular circulation. Since atropin has dangerous possibilities, particularly in eyes with vascular affections, its use is limited. But pilocarpine can be used without fear; it seems to exert a favorable effect in the retinal lesions of hypertension, especially when accompanied by local vasodilatation. In thrombosis of the central vein pilocarpin is particularly indicated, because of the frequent complication of glaucoma in this condition.

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